DO NOT CIRCULATE

Vol. 122, No. 1 July 1955

The Journal of NERVOUS AND MENTAL DISEASE

An Educational Journal of Neuropsychiatry, founded in 1874

UNIVERSITY OF MICHIGAN
JAN 3 1956
MEDICAL

MANAGING EDITOR
NOLAN D. C. LEWIS, M.D., *Princeton*

ASSOCIATE MANAGING EDITOR
A. N. FOXE, M.D., *New York*

ASSOCIATE EDITORS
PERCIVAL BAILEY, M.D., *Chicago*
KURT GOLDSTEIN, M.D., *New York*

Published monthly by
THE WILLIAMS & WILKINS COMPANY
BALTIMORE 2, MARYLAND

COPYRIGHT 1955, THE WILLIAMS & WILKINS COMPANY

Managing Editor
NOLAN D. C. LEWIS, M.D.
New Jersey Neuro-Psychiatric Institute
Box 1000
Princeton, New Jersey

Associate Managing Editor
A. N. FOXE, M.D., *New York*
Associate Editors
PERCIVAL BAILEY, M.D., *Chicago*
KURT GOLDSTEIN, M.D., *New York*

With the Collaboration of

A. AUSTREGESILO, M.D., *Rio de Janeiro, Brazil*
FRANCIS J. BRACELAND, M.D., *Hartford, Conn.*
LOUIS CASAMAJOR, M.D., *New York, N. Y.*
RUSSELL N. DEJONG, M.D., *Ann Arbor, Mich.*
JEAN DELAY, M.D., *Paris, France*
FREDERIC J. FARNELL, M.D., *New York, N. Y.*
JOHN F. FULTON, M.D., *New Haven, Conn.*
BERNARD GLUECK, M.D., *New York, N. Y.*
C. JUDSON HERRICK, Ph.D., *Chicago, Ill.*
MARION HINES, M.D., *Emory University, Ga.*
GORDON HOLMES, M.D., *London, England*
BEN KARPMAN, M.D., *Washington, D. C.*
WINFRED OVERHOLSER, M.D., *Washington, D. C.*
JEAN SAUCIER, M.D., *Montreal, Canada*
ALBERTO SEGUIN, M.D., *Lima, Peru*
E. A. SHARP, M.D., *Buffalo, N. Y.*
EDWARD A. STRECKER, M.D., *Philadelphia, Pa.*
L. VAN DER HORST, M.D., *Amsterdam, Holland*
HENRY R. VIETS, M.D., *Boston, Mass.*
ROBERT WARTENBERG, M.D., *San Francisco, Cal.*

PINEWOOD

DR. JOSEPH EPSTEIN
DR. LOUIS WENDER
Physicians in Charge

Dr. Walter A. Thompson, F.A.P.A., *Clinical Director*
Dr. Conrad K. Gale, *Senior Psychiatrist*
Dr. Jacob Schechner, *Senior Psychiatrist*

WESTCHESTER COUNTY **Katonah, N. Y.** **Tel: Katonah 4-0775**

A competent sixty bed psychiatric hospital offering an approved program for training residents in neuropsychiatry Psychoanalytically oriented. Patients receive individual treatment sessions and also group therapy. Electro shock therapy and its modifications available. Pharmacological therapies. Consulting staff of specialists. Monthly clinical conferences open to the profession.

N. Y. CITY OFFICES—BY APPOINTMENT
Dr. Wender—59 East 79 St.—Mon., Wed., Fri., BU 8-0580
Dr. Epstein—975 Park Ave.—Tues., Thurs., Sat., RH 4-3700

Department of Child Psychiatry
THE MENNINGER FOUNDATION

THE SOUTHARD SCHOOL

Intensive individual psychotherapy in a residential school, for children of elementary school age with emotional and behavior problems.

THE MENNINGER CHILDREN'S CLINIC

Outpatient psychiatric and neurologic evaluation and consultation for infants and children to eighteen years.

J. COTTER HIRSCHBERG, M.D., *Director*

Topeka, Kansas; Telephone 3-6494

Entered as second-class matter at the Post Office at Baltimore, Maryland. Subscription Price: One year, $14.00

HUGHLINGS JACKSON'S DOCTRINE OF APHASIA AND ITS SIGNIFICANCE TODAY[1]

WALTHER RIESE, M.D.

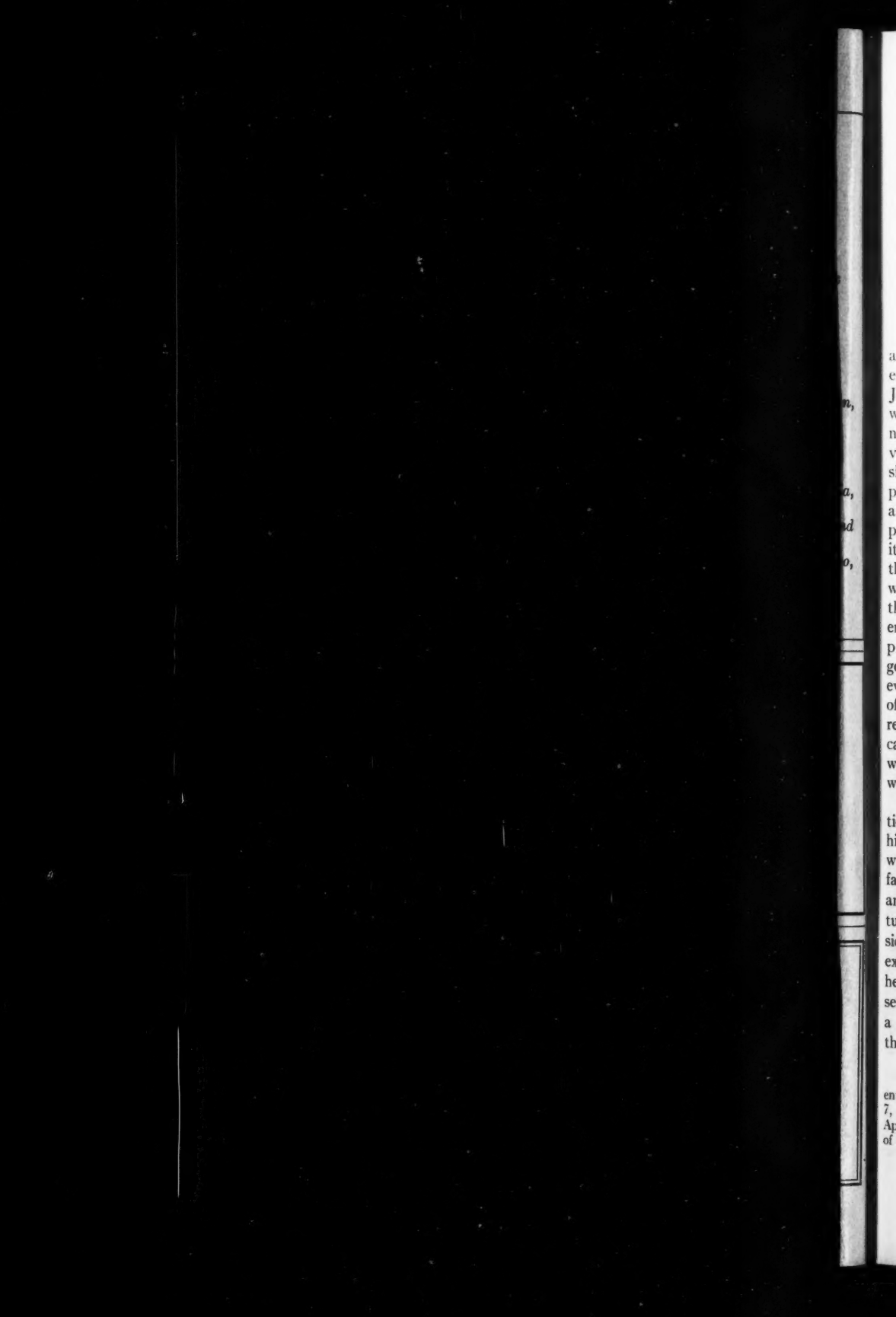
The Polemic Element in Jackson's Doctrine of Aphasia

There is to be found in Jackson's writings on aphasia an apparently insignificant polemic element. The fact is remarkable. As a rule, Jackson did not engage in polemics; the wealth of his own thought and experiences, the need, so intensely felt for developing and conveying his ideas to his contemporaries, the shortness of time left to a busy physician, perhaps also his kind and gentle nature, did not allow him to become entangled in more or less personal and sterile discussions. In this respect, it is significant that he confessed: "I cannot but think that my disagreement with M. Broca, as well as many of the differences of opinion on this subject, are to a great extent due to different ways of putting the same thing. . ." (5, II, p. 123). Before formulating his opinion divergent from that of Broca, he said: "I think the evidence I have collected goes to confirm many of his statements as to facts. . . ." That Broca remained himself critical in his approach and cannot be held responsible for the misuse to which the terms and concept of speech centers were subject, was shown by this writer (14).

I submit that Jackson assigned an exceptional importance to the polemic constituent of his doctrine of aphasia, brief as this constituent was. The target was Broca's assumption of a faculty of language, to which Jackson denied any existence. "Disease near the corpus striatum," he concluded, "produces defect of expression (by words, writing, signs, etc.), to a great extent, because this is the way out from the hemisphere to the organs which the will can set in motion" (5, II, p. 123). The formula was a masterstroke. It left to the brain the simple though still most important role of being instrumental in speech but denied to it any claim to serve as a center for language. There was embodied in this formula the principle of Jackson's interpretation of the nervous system as an organ of movements from the lowest and most automatic ones serving in respiration, to highly evolved, most voluntary, refined and educated movements serving in speech and conduct. The purely instrumental role of the nervous tissue in language was also expressed in the following formula: ". . . damage near the corpus striatum affects language and thought, not because any so-called faculty resides there (or anywhere, except in the whole brain or whole body) but because more or less of parts which help in making symbols are broken up" (5, II, p. 125). There is implied in the denial of the "faculties" and their residences, an anti-ontological attitude. From the beginning, the sensualistic tradition has been very conspicuous in the thought and writings of the British school of neurology. Again, it found its way into Jackson's writings: "There is no 'faculty' or 'power' of speech apart from words revived or revivable in propositions, any more than there is a 'faculty' of co-ordination of movements apart from movements represented in particular ways" (5, II, p. 160).

The doctrine of the faculties was medieval heritage. Though it was Galenic in origin, neither Aristotle nor Galen can be held responsible for any dogmatic and ontological use made of the faculties in their names at any period of medical history; this was shown recently by this writer (13). But at the time of the first systematic studies of aphasia, early investigators still labored under the influence of the doctrine of the faculties transmitted to them by the intellectual father of the speech centers, the once so popular F. J. Gall (9, 11), whose follower Bouillaud may be said to have opened the early history of aphasia as a chapter of medical history, almost 40 years before Broca delivered his famous lectures and demonstrations to several learned Paris societies (14).

[1] The text of this paper represents a major constituent of "A Workshop on Aphasia", given on May 6 and 7, 1955, by the author to the School of Clinical and Applied Psychology, Richmond Professional Institute of the College of William and Mary.



HUGHLINGS JACKSON'S DOCTRINE OF APHASIA AND ITS SIGNIFICANCE TODAY[1]

WALTHER RIESE, M.D.

The Polemic Element in Jackson's Doctrine of Aphasia

There is to be found in Jackson's writings on aphasia an apparently insignificant polemic element. The fact is remarkable. As a rule, Jackson did not engage in polemics; the wealth of his own thought and experiences, the need, so intensely felt for developing and conveying his ideas to his contemporaries, the shortness of time left to a busy physician, perhaps also his kind and gentle nature, did not allow him to become entangled in more or less personal and sterile discussions. In this respect, it is significant that he confessed: "I cannot but think that my disagreement with M. Broca, as well as many of the differences of opinion on this subject, are to a great extent due to different ways of putting the same thing. . ." (5, II, p. 123). Before formulating his opinion divergent from that of Broca, he said: "I think the evidence I have collected goes to confirm many of his statements as to facts. . . ." That Broca remained himself critical in his approach and cannot be held responsible for the misuse to which the terms and concept of speech centers were subject, was shown by this writer (14).

I submit that Jackson assigned an exceptional importance to the polemic constituent of his doctrine of aphasia, brief as this constituent was. The target was Broca's assumption of a faculty of language, to which Jackson denied any existence. "Disease near the corpus striatum," he concluded, "produces defect of expression (by words, writing, signs, etc.), to a great extent, because this is the way out from the hemisphere to the organs which the will can set in motion" (5, II, p. 123). The formula was a masterstroke. It left to the brain the simple though still most important role of being instrumental in speech but denied to it any claim to serve as a center for language. There was embodied in this formula the principle of Jackson's interpretation of the nervous system as an organ of movements from the lowest and most automatic ones serving in respiration, to highly evolved, most voluntary, refined and educated movements serving in speech and conduct. The purely instrumental role of the nervous tissue in language was also expressed in the following formula: ". . . damage near the corpus striatum affects language and thought, not because any so-called faculty resides there (or anywhere, except in the whole brain or whole body) but because more or less of parts which help in making symbols are broken up" (5, II, p. 125). There is implied in the denial of the "faculties" and their residences, an anti-ontological attitude. From the beginning, the sensualistic tradition has been very conspicuous in the thought and writings of the British school of neurology. Again, it found its way into Jackson's writings: "There is no 'faculty' or 'power' of speech apart from words revived or revivable in propositions, any more than there is a 'faculty' of co-ordination of movements apart from movements represented in particular ways" (5, II, p. 160).

The doctrine of the faculties was medieval heritage. Though it was Galenic in origin, neither Aristotle nor Galen can be held responsible for any dogmatic and ontological use made of the faculties in their names at any period of medical history; this was shown recently by this writer (13). But at the time of the first systematic studies of aphasia, early investigators still labored under the influence of the doctrine of the faculties transmitted to them by the intellectual father of the speech centers, the once so popular F. J. Gall (9, 11), whose follower Bouillaud may be said to have opened the early history of aphasia as a chapter of medical history, almost 40 years before Broca delivered his famous lectures and demonstrations to several learned Paris societies (14).

[1] The text of this paper represents a major constituent of "A Workshop on Aphasia", given on May 6 and 7, 1955, by the author to the School of Clinical and Applied Psychology, Richmond Professional Institute of the College of William and Mary.

The Basic Constituents of Jackson's Doctrine of Aphasia

I am inclined to believe that Jackson was inspired to conceive and to develop his doctrine of aphasia by an observation of stupendous simplicity, namely that an aphasic individual may be unable to repeat after the examiner such simple words as "yes" or "no", but shortly thereafter, and in the same session, may still be able to use the terms as replies, e.g., when asked whether he would like to go home. Similarly, he would say "Good-bye" when leaving the room, though he was found to be unable to repeat the same word after the examiner. Here was demonstrated, for the first time in the history of aphasia, *the flexibility of defects*. This meant the end of the famous doctrine of speech centers. It simply is not intelligible that a given speech defect is due to the destruction of a corresponding center when shortly thereafter the same defect is no longer demonstrable. Here we face on the smallest scale conceivable, the phenomenon of *recovery*. Logically extending the observation, we may conclude that the very fact of recovery speaks against the existence of speech centers. To allow other areas to "take over" the role of the destroyed speech centers would mean to postulate more than one center for the same performance; one may then ask why just two centers and why not consider the whole brain as one great center? But should the assumption of the vicarious function mean that after the destruction of the so-called speech centers the function would be accomplished by an as yet untrained area, the assumption would equally be detrimental to the hypothesis of specific centers for speech. Besides its fatal effect on the existence of speech centers, the flexibility of speech displayed by the aphasic individual serves to illustrate the two types of language distinguished by Jackson. There is implied in the patient's inability to repeat after the examiner the affection of Jackson's *voluntary* speech and in the preservation of the patient's ability to utter the same word under excitement or emotion, Jackson's *automatic* speech. It is in conformity with his law of evolution of nervous function and its reverse under abnormal conditions, i.e., dissolution, that the more voluntary functions suffer first and most severely, whereas the more automatic functions suffer last or not at all. He also considered the automatic language to be the inferior one. True speech, he said, is to propositionize. The unit of speech is a proposition, not simply an unrelated succession of words, but a relation in which the terms are modified by each other. It is propositional or superior speech which the aphasic individual has lost, but not the inferior, emotional, interjectional, in brief, automatic speech. One will have to bear in mind, however, that occasionally single words, such as "yes" or "no" may assume the roles and ranks of propositions if other words in relation are implied; this qualification has to be remembered in our attempts to analyze and to understand the true nature of speech lost or preserved in aphasic individuals; these may use recurrent or stock sentences, fragments or single words, as emotional or interjectional utterances, but also as propositions.

Jackson reached another definition of language of still greater philosophical scope, destined to assume an all-important place in the future work of one of his most eminent followers. Referring to the term of aphasia, given by Trousseau to defects as well as loss of speech, and referring to the preferable term of affection of speech (including defects and loss), but finally rejecting both terms as inappropriate and insufficient, Jackson stated: ". . .there is often a loss or defect in symbolizing relations of things in any way" (5, II, p. 159). It is in this vein that he laid the background for his definition of written language. "Written words," he said, "are symbols of symbols" (5, II, p. 163). Jackson probably did not know that Aristotle was his forerunner; as a rule, Jackson was more than generous in acknowledging his indebtedness to other authors, past and present. The second sentence in Aristotle's small treatise "On Interpretation" reads as follows: "Spoken words are the symbols of mental experience and written words are the symbols of spoken words."

Cerebral Dominance in Jackson's Doctrine of Aphasia

One of the major distinctions made by Jackson in his analysis of aphasia was that between a speechless and a wordless individual. The patient deprived of speech, i.e., of propositional language, still has words which occasionally he

is able to *utter* (though not to *say*); he retains automatic or emotional language. Since, as a rule, the left hemisphere is the seat of lesions in these cases, Jackson concluded that the function of the brain is dual, the left hemisphere being for voluntary speech and the right one for automatic speech, or, more correctly, because more faithful to the doctrine of concomitance that "nervous arrangements for words used in speech *lie chiefly* in the left half of the brain" and "that the nervous arrangements for words, used in understanding speech (and in other ways) lie in the right also" (5, II, p. 183). Retouching the suggestion of Dr. Moxon according to whom "the left side of the brain only is educated, although there is 'an organ of language' on each side. . ." (5, II, p. 124), Jackson concluded that "both sides are probably educated, but that the left is the one that begins to act, and that those educated utterances, which are in a sense involuntary, have become so by habit—i.e. frequent education—and may be the result of action on the right side only. . ." (5, II, p. 125). Jackson believed indeed that automatic revival in the right hemisphere always precedes voluntary reproduction on the left. "On the right half there is faint automatic reproduction of words *before* the stronger voluntary reproduction of the left" (5, II, pp. 137–138).

This writer cannot escape the impression that this conclusion remained in contrast with Jackson's historical dictum that "to locate the damage which destroys speech and to locate speech are two different things" (5, II, p. 130). The solution submitted by this writer is that in an intact brain each part cooperates in each function, though in an undetermined and always changing way and intensity. The loss of one hemisphere or of one of its parts leaves the remaining parts alone though, through their previous cooperation, not entirely unprepared to continue to function on a reduced scale and to serve in actions which remain fully integrated, i.e., total ones as long as there remains life even in the smallest fragment of an organized product of nature (12).

Inner Speech in Jackson's Doctrine of Aphasia

Jackson did not admit of any basic difference between external and internal speech. He reduced it to a difference of degree or that of a motor impulse or nascent movement from actual movement; psychically "faint" and "vivid", physically "slight" and "strong" nervous discharges. In internal speech, he said, there is *faint* excitation of those *central* sensori-motor processes which for external speech require to be strongly excited. "There is as much speech when I say 'Gold is yellow' to myself as there is when I say it aloud" (5, I, p. 85). To test his internal speech, we must ask an individual to write. A person could not write unless he spoke internally; but to speak meant to Jackson to propositionize. The aphasic patient does not write because he has no propositions to write, though he may write "in the sense of penmanship," (i.e., "copywriting, copy print into writing, make a meaningless succession of letters. . ." (5, II, pp. 162–164). Jackson believed the difference between external and internal speech to be a minor difference compared with that between the prior unconscious, or subconscious, and automatic reproduction of words and the sequent conscious and voluntary reproduction of words; the latter alone is speech, he repeatedly ascertained, either internal or external. There was implied in these statements Jackson's assumption that speech, internal and external, is preceded by an automatic and subconscious reproduction of words. Jackson's elimination of any fundamental difference between external and internal speech meant a great simplification of a most complicated and involved subject. The reader of Jackson's writings is impressed more than once with the author's continuous and intense striving for simplification, always made, however, with caution and great care to avoid oversimplification. But in his doctrine of aphasia simplification may have prevented him from exploring that large area which has become known to contemporary thought and experience as the syndrome of disordered inner speech. There is, however, one term in Jackson's terminology which seemed to be an analogue of inner speech as interpreted by contemporary writers, i.e., the term *pre-conception*. "In the voluntary operation," he said, "there is pre-conception; the operation is nascently done before it is actually done; . . . to 'will', to 'know', to 'intend', to 'try', to 'remember', . . . are different names for the subjective re-

production . . ." (5, II, pp. 199–200). Of course, Jackson was also familiar with paraphasia, one of the most significant symptoms of disordered inner speech. He ascribed it to a "hurried action in patients who have defect in speech . . ." (5, II, p. 194).

Inner Speech Today

Inner speech has become one of the most difficult and most confusing chapters of aphasia. In citing only three major contemporary interpretations, I am not too sure to do full justice to the authors from whom they are borrowed.

Monakow (7) understood inner speech to be the whole of latent engrams or verbal contents shaped during individual life and comprising an endless number of syntactically organized sentences. ("Much of our talk is made up of acquired forms") (5, II, p. 222). As far as the *expressive* component of inner speech is concerned, it is meant to cover the excitatory process immediately *preceding* the mobilization and actualization of sounds. As far as the *perceptive* component is concerned, it marks the stage immediately *succeeding* the reception of the acoustic or graphic image, the stage in which the semantic structure of the word is identified. Mobilization of sound itself as well as the reception of the spoken word belong to *external* speech.

In *disorders* of inner speech the patient fails to reach the *symbolic* significance of the words perceived and therefore to understand the meaning of spoken language, or, on the expressive side, to make the preliminary steps needed for diction, syntax and word finding. The results will be: wrong directions, mistakes in combination and sequences of syllables and letters, suffocation of sounds already evoked, their resounding, perseveration. Disordered inner speech is not a lasting loss of sounds, words or sentences, but rather a defect of *evocation*, an inability to bring to inner actualization those sound sequences which are needed at the moment and which precede expression.

Goldstein (3, p. 94) defined inner speech as

"the totality of processes and experiences which occur when we are going to express our thoughts, etc. in internal speech and when we perceive heard sounds as language. Inner speech is, on the one hand, in relation to the 'non-language mental processes', on the other hand, to the external instrumentalities (external speech). The first relation finds its expression in the organization of inner speech according to the 'inner speechform', in the selection of definite word categories by a special language, in its syntactical forms and in its grammatical structure, in the adaptation of the particular speech to the general and special situation in which it takes place, its dependence upon the environment, the listener, etc."

The characteristics of inner speech, he concluded, are determined by its relation to the structure of the non-language processes, that is to the processes of thinking but also to the external instrumentalities consisting of sounds, words, series of words, sentences, one form of naming and of understanding of language in familiar situations for which it has been conditioned, and finally of emotional utterances (3, p. 25). In disturbances of inner speech there is impairment of spontaneous speech and understanding, the latter less than the first; literal and verbal paraphasia; paralexia and particularly paragraphia, writing usually more affected than reading, dictation more than copying; disturbance of repetition, disturbance of spelling and of the capacity to combine letters into words. Not infrequent complications are amnesic aphasia and signs of purely acoustic aphasia (3, p. 231).

In none of the definitions mentioned so far is the communicative character of inner speech denied. An entirely different interpretation was given by Vigotsky (18). Inner speech, determined by him first negatively, is not simply preceding external speech or reproducing it in memory; it is not the inner aspect of external speech. The transition from inner to external speech does not represent a simple addition of the vocal aspect to silent speech. Positively speeking, inner speech represents, according to Vigotsky, a specific whole with its own laws and complicated relationships to other forms of speech activity. *Inner speech is speech for one self, external speech for others.* The most important characteristic of inner speech is its peculiar syntax. Compared with external speech, inner speech appears abrupt and incomplete. There are three sources for the abbreviation significant of inner speech, namely a tendency to predication, the reduction of the vocal aspect and certain fundamental semantic peculiarities, such as the preponderance of the *sense* of a word over its *meaning* (the meaning being only a constituent of the sense),

". . . A word taken separately in the dictionary has only one meaning. But this meaning is only a potentiality which gains its realization in speech; there it forms only one stone in the building of sense . . . . The sense of a word, says Paulhan, is a complex, moving, continually changing phenomenon which has different forms in different consciousnesses and in different situations. Therefore, the sense of a word is virtually unlimited. A word receives its sense from a sentence; the sentence itself receives its meaning from the context of a paragraph; the paragraph—from the context of the book; the book from the context of the whole work of an author . . ." (18, pp. 47–48).

agglutination and finally the merging of the senses of words into each other (the whole sense of a work contained in one name). In brief, transition from inner to external speech is a transformation of predicative and idiomatic speech into syntactic, developed speech intelligible to others.

With one exception (3, p. 94) contemporary neurologists have not taken advantage as yet of this new concept of inner speech and the insight it promises into the structure of speech disturbances resulting from brain lesions. (In his book Goldstein also submitted to a critical evaluation the divergent views of Piaget and their bearing on the development of inner speech in the human child.)

Language and Thought in Jackson's Doctrine of Aphasia

In order to understand Jackson's idea on the interrelation of language and thought, one must bear in mind the distinction he made (in 1875) between "a voluntary internal reproduction of words in new and propositional forms (as occurs when we write)" and "an automatic internal reproduction of words in old and acquired forms, or in forms given us, as when we receive and understand words in propositions spoken to us" (5, I, p. 65). The same idea was expressed in the following quotation from a paper published a few years earlier: ". . . in the process which leads to outward speech—not necessarily outward, of course, let us say, to propositions—there is a double revival of words—automatic revival prior to voluntary revival—and that the *automatic* revival occurs first, and on the *right*, and the voluntary revival afterwards on the *left*, side of the brain" (5, II, p. 227).

Reviewing the evidences in the text and trying to shape them into a whole likely to reflect as faithfully as possible Jackson's thought on the subject, I reach the following conclusion: If thought be understood to be synonymous with propositionizing, the aphasic person, deprived of propositional speech, is by this same reason affected in his thinking. But inasmuch as he still has in automatic forms all the words he ever had, "cases of loss of speech do not give proof that thought is possible without words" (5, II, p. 206). The aphasic individual can still think. "He will be lame in his thinking, because, not being able to revive words (to speak to himself), he will not be able to register new and complex experiences of things" (5, II, p. 131 n.), the implication precisely being that "in the process which leads to propositions, there is a double revival of words—automatic revival prior to voluntary revival—and that the *automatic* revival occurs first and on the *right*, and the voluntary revival afterwards, on the *left*, side of the brain" (5, II, p. 227). The idea of a double revival of words took shape in Jackson's mind when he made the observation that an aphasic individual, though unable to speak, may still be able to understand; again, the implications being, 1) that the left half of his brain was affected and 2) that the process of understanding speech of others can be interpreted simply as automatically "receiving speech," the listener being the "victim" of the speaker. It would be unfair to ignore Jackson's qualifying remark that the expression "receiving speech of others" should not be taken literally. "We understand a speaker," he said, "because he arouses our words" (5, II, p. 206 n.). Retouching the sharpness of cerebral localization implied in these statements, he concluded (in 1874) ". . . the right is the half of the brain for the automatic use of words, the left the half for both the automatic and the voluntary use" (5, II, p. 130 n.). Jackson referred to the expression of the left as the "leading" half for words (speech) as to an expression "formerly used" by him. In a recent publication (8) this writer cited the conclusion reached by "contemporary students of *aphasia* that thought and language are neither identical nor parallel, though closely related to one another." I quoted Pick, according to whom "verbalization is, as a rule, preceded by a scheme or design of pure thought. But these two stages,

leading from thought to speech, are not always sharply demarcated; at times verbalization taking place, while thought is still being organized. Therefore, the more verbalized thought, the greater the effect of aphasia on thought. . . . But verbalization affects clearness, differentiation, and logical sequence of thought in different individuals in different ways."

An Early Follower? Freud's Conception of Aphasia

Introductory remarks

Freud's publication on aphasia (2a) almost entirely escaped the attention of his contemporaries, though in this small book (107 pages) the author discussed basic problems of aphasia and cerebral localization. According to Jones (6), Goldstein was the *only* one to cite it. It has never been analyzed and reevaluated in the light of recent knowledge. One of the most remarkable and distinctive characteristics of this book is that it was written by an author, who, as he confessed himself in the very beginning, had no personal observation to offer. This makes the book a rare and brilliant piece of medical *thought*. It can be divided into two sections. The first is polemic and aims mainly at the refutation of the famous, and at that time still most powerful doctrine of speech centers. One follows with greatest intellectual pleasure the author's intentions and method to meet and to destroy the thesis of his opponents on its own ground, exposing the *physiological* interpretations which unwittingly the adherents to the doctrine of speech centers introduced into their concepts, intended indeed to be purely *anatomical* ones. In the second section of the book Freud's own views were outlined.

Freud's Interpretation and Classification of Aphasia

Freud considered all cases of aphasia as the result of interruptions of associations (2a, p. 69). He said that the word (2a, p. 79) represents a complicated process of associations in which visual, acoustic and kinesthetic elements participate. It gains its meaning or symbolic nature through association with the representation of an object, which is in itself a complex of associations of the most diverse, visual, acoustic, tactile, cinesthetic and other representations. Using clinical experiences of speech defects, Freud advanced the thesis that *the representation of the word, more specifically, its acoustic element or sound, is linked with the representation of the object* (2a, p. 80).

On the ground of this thesis, he distinguished two orders of speech defects:

1) Aphasia of the first order or *verbal aphasia*, in which the associations between the various elements of the representation of the word (Wortvorstellung) are interrupted;

2) Aphasia of the second order or *symbolic aphasia*, in which the association between the word-representation and object-representation (Objektvorstellung) is severed.

Disorders in recognition he called *agnosia*, a term which has since become technical and irreplaceable. Agnosia may lead to aphasia of the third order or *agnostic aphasia*.

Freud's Conception of Localization of Speech

Freud strongly rejected the assumption of isolated cortical centers separated by functionless areas, these centers being supposed to store memory images or other psychical elements. He also rejected the assumption of subcortical fiber tracts providing exclusively for the association of these hypothetical centers. In fact, he did not distinguish between representations and associations; subsequently, he denied to the former any well-defined cortical area. He conceived the speech area as a continuous cortical region within which the associations and transmissions underlying the speech functions are taking place; they are of a complexity beyond comprehension, he concluded. The critical area of speech extends, he believed, from the cortical endings of the sensory nerves to the motor areas to which he left their cerebral localizations. These endings surround the speech area proper. The more centrally a given lesion tends to be placed within this area, the less will the effect be in terms of loss of one of the constituent elements of speech associations, and the more it will be in terms of purely physiological factors presiding over speech regardless of the site of lesions. Freud conceived these factors as basic properties of a cerebral mechanism subserving associations (2a, p. 91). They have been described

and analyzed by this writer as "dynamics in brain lesions" (unpublished address read at the Chestnut Lodge, Rockville, Maryland, February 10, 1953).

Though Freud stressed the difference between cerebral localization of *functions* (which he denied to any highly evolved psychic act such as language) and the cerebral localization of *lesions* (which, of course, he admitted), the contemporary reader cannot escape the impression that Freud did not completely liberate himself from a neuroanatomical conception of aphasia. In fact, he conceived aphasia as disturbed conduction, an ambiguous term, used sometimes in an anatomical, sometimes in a physiological sense. Nor could Freud renounce diagrams. Occasionally he went so far as to expect the explanation of individual variations of asphasia from a better knowledge of the anatomy of (crossed) connexions (2a, pp. 67–68).

Nevertheless, Freud confessed that it was Hughlings Jackson "on whose views I have based almost all the arguments which I have advanced in refuting the localizatory theory of the aphasias." It is true that Freud too was inclined to explain the recurrent utterances of aphasic patients "by their intensity if they happen at a moment of great inner excitement." "I remember having twice been in danger of my life," he reported, "and each time the awareness of the danger occurred to me quite suddenly. On both occasions I felt 'This is the end,' and while otherwise my inner language proceeds with only indistinct sound images and slight lip movements, in these situations of danger I heard these words as if somebody was shouting them into my ears, and at the same time I saw them as if they were printed on a piece of paper floating in the air" (2, p. 62). But this writer failed to rediscover in Freud's own views on aphasia the principles of evolution and dissolution which are the pillars on which rests Jackson's doctrine of aphasia.

Freud's treatise on aphasia appeared in 1891. As early as 1874, Jackson (5, II, p. 129) had formulated his views on aphasia which remained basically the same in his later publications. These views aprang from his definition of speech as the power to propositionize. Even at this early moment he was able to quote in a footnote the following passage from a paper published by him as early as 1868:

> "It is not enough to say that speech consists of words. It consists of words *referring to one another in a particular manner;* and without a proper interrelation of its parts a verbal utterance would be a mere succession of names embodying no proposition" (5, II, p. 130).

Ten years later he again stated (5, II, p. 159):

> "To speak is not simply to utter words, it is to propositionize."

He now defined a proposition in a much more precise manner as

> ". . . such a relation of words that it makes one new meaning, not by a mere addition of what we call the separate meanings of the several words; the terms in a proposition are modified by each other. Single words are meaningless and so is any unrelated succession of words."

The conclusion he reached reads like a manifesto: "The unit of speech is a proposition." One wonders why Freud, though an avowed adherent of Jackson, did neither mention nor consider these views and statements. One reason may be found in the fact that the first of the two papers by Jackson, as so many others, was published in a periodical which, at that time, may not have been easily accessible to readers on the European Continent. Be that as it may, Freud's interpretation of aphasia still centered mainly around the elements of speech, i.e., words and their associations. I submit that in this respect it remained behind Jackson's views which sprang from his concept of speech as an organized whole whose parts are interdependent, each element owing its position and its significance to its relation to every other element and to the whole. Since this view reflects the rational interpretation of an organism, one might call it an organismic view of language. It remains even open to doubt whether or not Freud left intact the strictly *functional* design of the Jacksonian evolution, or whether from the onset he substituted a *genetic* principle for an evolutionary thought to which chronological or historical elements were entirely foreign. Freud's general interpretation of aphasia as an "earlier" level of functioning (2, p. XII) seems to justify this

assumption. Similarly, he explained speech disorders in the polyglot through the survival of the earlier acquired language. Since then, observations have been accumulated demonstrating exceptions to this rule and calling for a strictly individual analysis of each case. It remains significant that Jackson warned his readers that evolutionism is not synonymous with Darwinism and that in contrast Freud's final thought and writings assumed an unmistakable Darwinian character. This comment is not out of place since rapid readers of the treatise on aphasia are inclined to indulge in an undue glorification of *all* views offered in this treatise by its eminent author.

Therefore, it may not be surprising that Freud did not adopt the Jacksonian dynamic principles for the interpretation of cerebral *paralysis*. He explained its clinical manifestations by anatomical arrangements, though he also did say that the cortico-fugal tracts have a much stronger influence on the upper than on the lower extremities, and though in a general way he objected to a rigid equation of the degree of tissue damage with the resulting functional deficiencies. Freud's "Infantile Cerebrallaehmung" (1), published in 1897, i.e., six years after his treatise "On aphasia", comprised 327 pages. Neither in the chapter on patho-physiology nor in the bibliography carrying no less than 416 numbers was the work of Jackson mentioned. Nor did Jackson's name recur in the special chapter devoted to Freud's work on paralysis in Spehlmann's recent synthesizing review of Freud's neurological contributions (17). But why did Freud who acknowledged his debt to Hughlings Jackson neither make full use of Jackson's doctrine of evolution and dissolution, in 1891, when discussing aphasia nor in 1893, when interpreting cerebral paralysis? Outstanding and independent minds often refuse, and rightly so, to shape their own discoveries in the terms of others whose thoughts at first approach do not seem to differ from their own thoughts, and whose influence may have reached them more than once. Stengel, who translated Freud's treatise on aphasia and who was striving earnestly and successfully to identify in this treatise anticipations of Freud's final thought and work, recalled Freud's interpretation of paraphasia, i.e., the mistaken use of words. The critical passage, Stengel said, reads like a prelude to the chapter on errors and slips of the tongue in "Psychopathology of Everyday Life." What in my opinion is of greatest significance for the understanding of the dynamics in brain lesions, was Freud's conclusion that his conception of paraphasia "as a purely functional symptom, a sign of reduced efficiency of the apparatus of speech associations . . . does not exclude that it may occur in most typical form as an organic focal symptom" (2, p. 13). Here, then, was adopted for the first time the view that *the organic genesis of a given symptom does not interfere with the undeniable observation that the same symptom remains subject to the physiological and psychological laws presiding over the appearance of symptoms at large, due or not to destroying lesions.* It has not been realized as yet that this conclusion meant the end of one of the most paralyzing and sterile dialectics under which those neurologists have to labor, who strive for insight into neurodynamics rather than for classification and rapid diagnosis, namely the dialectics between "organic" and "functional".

Rediscovery and Reshaping: Henry Head on Aphasia

Henry Head's work on aphasia, comprising 2 volumes and 949 pages, emerged as a truly monumental elaboration and continuation of Jackson's doctrine. At the time of the publication of Head's work, it had become conventional to classify speech defects resulting from brain lesions as "motor" or "sensory", to cite but the two major groups. The polemic part of Head's work was directed against this classification and the purely physiological analysis of aphasia which this classification implied. Underlying this classification, he said,

> "is the prevalent fallacy that, when a highly complex process breaks up, its component elements are betrayed by the loss of function or negative effect. But we cannot even analyze normal speech strictly in terms of motion, or sensation; most acts depend on both these factors for perfect execution. For the use of language is based on integrated functions, standing higher in the neural hierarchy than motion or sensation, and, when it is disturbed, the clinical manifestations appear in terms of these complex psychical processes; they cannot be classed under any physiological categories, motor or sensory, nor even under such headings as visual and auditory . . ." (4, pp. 202–203).

"The more complete such cases are examined the less they do conform to the current views of the nature of aphasia and the less easy is it to explain the clinical manifestations. If this is true with 'motor' defects of speech, the difficulties become multiplied indefinitely in the so-called 'sensory' forms . . ." (4, p. 204).

The aphasic patient, he concluded,

"had lost the capacity to perform acts not usually comprised under the use of language, but profoundly dependent on the power of internal verbalization . . ." (4, p. 203).

Strongly supporting Jackson's observations on the flexibility of the speech defects encountered in these patients, more particularly the influence of emotion, Head concluded that

"the various mental activities, interrupted during acts of symbolic formulation and expression, can frequently be exercised in a normal manner during some other form of behavior. Thus the patient may have profound difficulty in manipulating visual images at will for the purposes of verbal expression, although he can employ them with ease in other modes of thinking. It is not images as such that are disturbed, but images used in a certain manner directed towards a definite end" (4, pp. 210–211).

The power of understanding what is said to the patient "depends on the way in which the words are presented" (4, p. 205). He also stressed the metaphorical expression so frequently employed by patients suffering from naming defects; these metaphorical expressions revealed the patient's ability to describe accurately colors which the patient was unable to name.

"Less severe destruction of speech disturbs the use of words in such a way, that the higher and more abstract the 'proposition' the more likely is the patient to fail not only in the emission of a correct verbal equivalent, but in the recognition within himself of the full value of the 'proposition' (4, p. 209).

But Head objected to Jackson's term "propositionizing." Under this "uncouth word" was included what Head understood by "symbolic formulation and expression."

"I have combined," he concluded, "under the general heading of 'symbolic formulation and expression', the disorders of language produced by a unilateral lesion of the brain, because in the majority of instances the gravest disturbance is shown in the use of such symbols as words and figures. But any form of mental behaviour is liable to suffer which demands perfect reproduction and use of any symbol between its initiation and fulfilment. I do not believe that it is possible to include within one categorical definition all those activities which experience shows to be affected; and yet from a physiological point of view they form a group of defects as definite as those of sensation" (4, p. 210).

Thus was reached for the second time in the history of human thought as it is embodied in our subject, the stand taken by Aristotle.

". . . any act of mental expression, which demands symbolic formulation, tends to be defective and the higher its propositional value the greater difficulty will it present . . .

Any modification of the task, which lessens the necessity for symbolic representation, will render its performance easier . . ." (4, p. 212).

Head designed special tests by which the patient's disabilities were graded from pure matching or imitating, i.e., purely automatic performances to those implying symbolic meaning and inner verbalization (from copying printed matter in capital letters to copying print in cursive handwriting; from counting and saying the days of the week or the months of the year to recognizing the significance of any single number, day or month; from setting the hands of the clock at 3:40 to placing them at 20 minutes to 4, the task involving the symbolic value of the hands but also of their relative position on the face of the clock; from matching the examiner's movements reflected in a mirror to reflecting them when sitting face to face with him). It seems that facts are registered correctly but that the aphasic patient cannot relate them to one another. Finally, the ultimate intention of the symbol may be disturbed and the patients lack the clear conception of the goal of an action they are asked to perform—they may not know how to change clocks—whether put forward or back, when passing to summertime; how to solve simple arithmetic tests, though the direct significance of figures is not affected; how to play such games as billiards and cards, implying the difficulty in formulating beforehand the aim or goal of some particular act.

Kurt Goldstein on Aphasia

From Jackson's inability to propositionize, a direct road leads to Henry Head's conception of aphasia as a disorder in symbolic thinking and expression. In this definition the most characteristic and most noble function of lan-

guage is expressed, namely, being *representative* of inner states, needs, and concepts. Among the types of aphasia distinguished by Henry Head the semantic defect assumes the most general character. In this disorder of symbolic formulation, he said, the fault is essentially a want of recognition of relative significance and intention. Everything tends to be appreciated in detail, but the general significance is lacking. One might raise the question whether this variety can still be classified with speech defects. Henry Head himself speaks of a loss of power to appreciate or to formulate the logical conclusion of a train of thought or action. Perhaps less fortunate was Pierre Marie's famous attempt to interpret aphasia proper as a lack of intelligence, the term being too vague and requiring qualification, but conveying nevertheless a most general behavior. It is not so well known that similar interpretations of aphasia had been made previously by Trousseau, who wrote as early as 1868 that the aphasic has lost at the same time, though to various degrees, the memory of words, of the acts by means of which words are articulated, and intelligence. Leaving alone other contemporary attempts to interpret aphasia in the light of a disorder of basic functions (Bouman and Gruenbaum, van Woerkom), we finally quote Goldstein. Speech, he said, is one of the essential characteristics of human nature, inasmuch as it is tied to man's highest capacity, the capacity of abstract behavior. The latter is also called by him the categorical behavior, or the objectifying attitude, or even voluntary behavior. In the abstract attitude we are not directed toward an individual object but toward a category, of which that object represents itself only as an accidental example and representative of that category. To this behavior Goldstein opposed the immediate concrete behavioral attitude, a more primitive one, in which the action is directly determined by momentary sense impressions, in which we are directed rather toward the concrete thing in its particular uniqueness, and in which words are used less as representative of categories than as individual properties, which like other properties belong to the object in question. Due to the cortical lesion, the (amnesic) aphasic is able to assume only the more concrete, the more realistic attitude. Not only speech but also his general behavior is characterized by a greater concreteness (which Goldstein sees in the predominance of acting over thinking) (10, 16).

There can be no doubt that Goldstein's interpretation of aphasia is the most general one ever conceived. He believes speech defects resulting from brain lesions reflect in a particular field the basic disturbances found in brain lesions at large, such as, the deficient distinction in a given situation between essential and nonessential factors, the reduced understanding of the situation as a whole, the inability to shift from one attitude to another, and subsequently, the rigidity so significant of brain injured persons, their inability to detach themselves from a given stimulus and to react simultaneously to two different stimuli, their inability to make in a general way selections, and subsequently, their loss of freedom, the priority given by them to purely formal and external rather than inner principles of classification and order, their anxiety when facing emptiness, the incomplete demarcation of the individual from the surrounding world. It is Goldstein's conviction that according to the functions tested the same basic disorder may explain aphasia, apraxia, agnosia, lack of orientation and attention, dementia, and even apparent lack of emotional response. In still more general terms, the basic disorder is defined by him as a more concrete attitude and opposed to a more abstract or categorical one. Goldstein was impressed by the answer given by patients suffering from amnesic aphasia who named the color of a given object, not simply blue, but sky-blue, not simply red, but blood-red, and who were unable to sort out woolen threads according to the category of the name. Goldstein concluded that in these patients the name of the color was in indissoluble union with the object and that the patient was no longer able to reach the name by abstract and purely conceptual thought. The world of the brain-injured patient, he believed, has become a more individualized and a more intuitive one; the patient falling back to a more concrete attitude has left the highest and specifically human level of abstract thought.

The Writer's Approach

At an early period of my investigations (15), I have been attracted by those symbolic ex-

pressions which represent the functions preserved or, in Jacksonian terms, the so-called positive symptoms in aphasia, i.e., the surviving behavior best fit to meet the requirements arising from the new condition or emergency (Jackson's *survival of the fittest*, the only Darwinian term he used in his writings and obviously teleological in meaning). Occasionally, these patients produce neologisms, evidently to compensate their naming defects; these neologisms impress the listener by their descriptive and figurative power though, of course, they lack basic elements of artistic production, namely, planning, effort and training. Still, in one of my observations, that of a highly educated scientist and humanitarian, a new style appeared during his aphasia after a brief initial period of complete loss of speech; language, though still scanty and slow, became formal, solemn, poetical, dramatic, pathetic, and "Shakespearean", the patient frequently using quotations. What the brain injury brought to the fore was that element in his nature which disease could not *destroy*, but rather *release*. More than once in my investigations, I reached the conclusion that disease may occasionally reveal, though in a distorted fashion, what is great and noble in man's nature. What may be the "simple" to others, was the "complex" to our patient, and vice versa. There was still passage from the more "voluntary" to the more "automatic," the new terms being expressed with rapidity and fluency, and neither accompanied nor preceded by any active effort or intention. In so far, there was still dissolution of speech and passage from the more "voluntary" to the more "automatic". *Jackson's law of dissolution still holds true, but it must be interpreted and applied individually according to the meaning to be given to the term of the law in a particular case.* It is obvious that the *principle of individual dissolution* here enunciated has far-reaching practical implications in any kind of *testing*. Recently, I analyzed the auto-observation left by an eminent nineteenth century French scientist who retained the power to plan and to organize his lectures while affected by aphasia (8). The analysis of speech disturbances resulting from brain lesions in highly educated and outstanding people promises, it seems, greater insight into certain neuro- and psychodynamics than the analysis of aphasia occurring in the so-called norm-average, whose terminology, as a rule, is comparatively reduced, whose abstracting power and self-observing ability remain undeveloped, though the distinction here made can claim no more than statistical truth and may be denied by individual cases. With these restrictions in mind, I submit the English though abbreviated version of Forel's auto-observation of aphasia. [The original German text was brought to my knowledge by E. Bay (Heidelberg); the translation was undertaken with the assistance of Dr. G. E. Arrington (Richmond, Virgnina).]

Addendum

A. Forel's auto-observation of aphasia

At the age of 64, A. Forel suffered a stroke. It was preceded by a period of intellectual overwork and inner unrest due to the manifold preparations required by his plan to undertake a journey to certain tropical areas in Africa and Asia. He had just concluded an important and difficult investigation on ants and was very tired. While dictating a paper on the methods and meaning of comparative psychology, he experienced paraesthesia in his right arm and difficulties in finding the right terms. He thus felt unable to develop a coherent thought. While searching in the dictionary for terms with which he used to be very familiar, he forgot what he intended to say. Some words were simply omitted in the dictated text. The next day he was still able to express his thought in conversation, though with greatest difficulty. About a week later, he went to a meeting which he had promised to attend, but when he tried to address the audience, he again failed to find the right words, became confused, excited and depressed. This incident was later confirmed by some one who was in the audience and who also asserted that Forel did not say anything wrong. On his way home he was extremely tired and slightly somnolent. The next morning, after a good rest, he suddenly fell to the ground. He was not rendered unconscious; very soon he realized that his right arm and face and to a lesser degree his right leg were paralyzed. His speech was unintelligible (Forel was decidedly right-handed). He realized with great concern his condition and its unavoidable and detrimental effect on his life, economic situation, scientific projects and manifold obligations. Disregarding his condi-

tion and the protests made by his wife, he continued dictating letters in the morning and afternoon, but his secretary could not understand him too well, particularly in the afternoon. The diagnosis of apoplexy (thrombosis) was made by two friends, both outstanding neurologists, who forbade intellectual activity and dictation.

"Now my only choice was to accept my fate philosophically. Nevertheless—thoughts are free—in spite of my best intentions to obey my colleagues, my mind continued to work permanently and I considered every angle of the serious effects of the situation, whether to give up my journey and whether to care for the immediate and most urgent arrangements in case of a recurrence of the stroke. I feared that my condition would worsen and that I would suffer a relapse and that my mind would completely deteriorate; therefore, there was no time to lose. As a result, I annoyed my poor wife, against medical advice with all kinds of orders and notes on my bequests and other things which alarmed me."

A few days later, in order to divert himself, he wrote a poem, imitating a type of contemporary poetry about Darwinism. He described in his poem his imminent mental deterioration as the final stage of phylogenesis leading from the lowest to the highest level, namely that of the scientist, who through a stroke again regresses to a state of dementia. He confessed that he had not the slightest gift for poetry, but that he knew the metre. In fact, while he was able to create the rhyme, he was unable, in spite of repeated efforts, to produce a consistent metre; there were also other mistakes.

Soon Forel reassumed his scientific investigations, but according to his own opinion expressed in his retrospective report (published three years after his attack) his first "post-apoplectic" writings were definitely weak. About six weeks after his stroke improvement was definite, though not very marked. The mild paralysis of the right leg was no longer present, and more and more the speech disturbances assumed the type of simple articulatory defects (dysarthria) and some difficulty in evocation. Motion returned to his right arm, and he was able to write his revised will with his right hand. Nevertheless, he now tried to learn to write with his left hand. He had the greatest difficulties in carrying out simple calculations. He realized these difficulties to their full extent, but he could not avoid them. Similar mistakes were made in other mental operations. He omitted letters in writing, he forgot many words well known to him and used wrong expressions in dictating. He forgot actions just performed, thoughts and words just used. At that time he corrected his paper on "Methods and Meaning of Comparative Psychology" and the metre in his poem. He also began to study the material on ants collected by another scientist in Dutch-India. He trained himself in speaking and working slowly, scanning the syllables as stutterers are taught to do. About 4 months after the stroke, he attended in Zurich the meeting of the International Association for Medical Psychology and Psychotherapy; he was able to follow the discussions in which he also participated, though only occasionally and hesitatingly. His speech was as deficient in German as it was in French. Shortly thereafter the paralysis of his right arm became worse, affecting particularly thumb, index finger and middle finger. For several months he became unable to use the magnifying glass which he so badly needed for his studies on ants; nor was he able to write any more or to ride a bicycle. He made a tremendous effort to adjust himself to the new condition, writing with his left hand, using both hands and teeth for all kinds of tricks in dressing, undressing, and for physical work at large. He felt that he would have deteriorated mentally had he given up. He was rewarded by his success. But he was very positive in stating that the success was neither due to the restitution of destroyed tissue nor to vicarious function of other neurons. The defect in articulation and evocation remained stationary. But he finally succeeded in pronouncing every word, provided his speech was slow or scanning; certain consonants alone remained difficult to pronounce. Reading was not intact; he misinterpreted words and entire sentences. Interruptions disrupted his thought when dictating, and he would forget the beginning of long sentences. He made mistakes in reading the thermometer and did not grasp the meaning of a changed address on the envelope of a letter addressed to his son living in another town. At the time this auto-observation was made and published, Forel reported on 16 scientific papers on ants published after his stroke with two more in print; 20 more articles deal-

ing with other topics were published during the same time in newspapers and periodicals. To these must be added a popular edition of his "Sexual Problem", new editions of his "Mental Hygiene", "Brain and Mind", "Life and Death". About three years after his stroke, his pamphlet on "The United States of the Earth" was published. He remained convinced that in a way his post-apoplectic writings were better, at least more cautiously written than his previous ones, due to the greater slowness forced upon him and the frequent revisions and corrections which the restricted and less tense mode of life and work allowed him to make. This compensated in part his previous hasty mode of intellectual activity. He did not deny "great gaps and errors" in the mechanics of thought. He had to undergo two operations for glaucoma and finally to give up the descriptive work on ants.

"The worst thing for an old brain," he said, "would be to give up either voluntarily or involuntarily, its usual work, as for a young brain, the worst thing would be not to learn how to work . . . To live a little longer with nothing to do is purposeless neither for one self and for the community. It is much better that an old body and brain be used actively as long as possible."

He died at the age of 83, 19 years after his stroke.

BIBLIOGRAPHY

1. Freud, S.: Die infantile Cerebrallaehmung in *Spezielle Pathologie und Therapie* (1893), herausgegeben von H. Nothnagel, IX. Band, III. Teil; Alfred Hoelder, Wien, 1901.
2. Freud, S.: *On Aphasia.* A critical study by Dr. Sigmund Freud. New York: International University Press, Inc., Translated by E. Stengel, 1953.

2a. Freud, S.: *Zur Auffassung der Aphasien.* Eine kritische Studie von Sigmund Freud. Leipzig und Wien: Franz Deuticke, 1891.

3. Goldstein, K.: *Language and Language Disturbances. Aphasic Symptom Complexes and their Significance for Medicine and Theory of Language.* New York: Grune & Stratton, 1948.
4. Head, H.: *Aphasia and Kindred Disorders of Speech.* Cambridge: University Press, 1926.
5. Jackson, J. H.: *Selected Writings of John Hughlings Jackson.* Edited by James Taylor, Gordon Holmes and F. M. R. Walshe. London: Hodder and Stoughton, 1931–1932.
6. Jones, E.: *The Life and Work of Sigmund Freud.* Vol. I. New York: Basic Books, Inc., 1953.
7. Monakow, C. von: *Die Lokalisation im Grosshirn und der Abbau der Funktion durch kortikale Herde.* Wiesbaden: J. F. Bergmann, 1914.
8. Riese, W.: Auto-observation of aphasia. Reported by an eminent nineteenth-century medical scientist. *Bull. Hist. Med., 28:* 237–242, 1954.
9. Riese, W.: F. J. Gall et le problème des localisations cérébrales. *Hyg. ment., 31:* 105–136, 1936.
10. Riese, W.: Kurt Goldstein's Stellung in der Geschichte der Neurologie. Versuch einer Wuerdigung aus Anlass seines 70. Geburtstages. *Schweiz. Arch. f. Neurol. u. Psychiat., 62:* 1–10, 1948.
11. Riese, W.: Les discussions du problème des localisations cérébrales dans les sociétés savantes du dix-neuvième siècle et leurs rapports avec des vues contemporaines. *Hyg. ment., 31:* 137–158, 1936.
12. Riese, W.: *Principles of Neurology in the Light of History and Their Present Use.* New York: Nervous and Mental Disease Monographs No. 80, 1950.
13. Riese, W.: *The Conception of Disease, Its History, Its Versions and Its Nature.* New York: Philosophical Library, 1953.
14. Riese, W.: The early history of aphasia. *Bull. Hist. Med., 21:* 322–324, 1947.
15. Riese, W.: Ueber den Entstehungsmechanismus der Wortneubildungen bei Amnestisch-Aphasischen. *Monatsschrift f. Psychiat. u. Neurol., 68:* 507–514, 1928.
16. Riese, W., and Hoff, E. C.: A history of the doctrine of cerebral localization. Methods and main results. *J. Hist. Med. and Allied Sc., 6:* 439–470, 1951.
17. Spehlmann, R.: *Sigmund Freud's neurologische Schriften.* Eine Untersuchung zur Vorgeschichte der Psychoanalyse. Berlin, Goettingen, Heidelberg: Springer, 1953.
18. Vigotsky, L. T.: Thought and speech. *Psychiatry, 2:* 29–52, 1939.

MEDICAL COLLEGE OF VIRGINIA
RICHMOND, VIRGINIA

CAPILLARY STRUCTURE: ITS RELATION TO PSYCHIATRIC DIAGNOSIS AND MORPHOLOGY[1]

NANCY WERTHEIMER, PH.D., AND MICHAEL WERTHEIMER, PH.D.

Hauptmann and Myerson (5) reported in 1948 that capillary structure, as observed at the nailfold, differentiated characteristically between groups of normals, paranoid schizophrenics, and non-paranoid schizophrenics. This study was designed to test, among other things, whether or not such differences in capillary structure persist when somatotype is held constant.

A second purpose was to determine whether or not certain functional measures are related to capillary structure. If they are, this would imply that future studies relating these functional measures to a disease syndrome such as schizophrenia should take into account the role of the morphological variable as a partial determinant of the results.

Third, as a necessary preliminary step in the study, an attempt was made to set up a standard for the measurement of capillary structure and to investigate just what the structural differences observed might mean about the general structure and function of the individual.

APPARATUS AND PROCEDURE

A schematic cross-section of the skin is shown in figure 1A. The two main layers, the epidermis and corium, originate from ectodermal and mesodermal tissue, respectively. The vessels of the subpapillary plexus (the arterial vessels feeding and the venous vessels collecting blood from the skin capillaries) lie at the base of the corium (tunica propria) and run more or less horizontally to the skin surface. The capillary loops, or papillary capillaries, grow vertically up from these vessels into the corpus papillare, each loop growing into one of the corium papillae.

Looking down through the skin, it would be difficult to determine the shape of one of these vertical capillary loops since little more than the top bend of the loop would be visible. In the fingernail bed, however, the most distal row of capillary loops, those just below the cuticle, are oriented more or less horizontally to the skin surface and are therefore more easily studied (fig. 1B).

For observation of these capillaries, a standard microscope was used with a 5× eyepiece and a 10× objective. The skin just over the nailfold was washed with 70 per cent alcohol, then lightly painted with cedar oil to make the skin surface more transparent. The nailfold of a given finger was positioned directly under the lens and held in this position by one of the examiner's hands; his other hand was used to focus the microscope. Focused on the finger at an angle to about 30° with the horizontal was a strong lateral light equipped with a green filter. As the examiner looked through the microscope, the finger was moved about until the entire terminal row of capillary loops had been observed as well as the region about 1 mm. or so proximal to this end row. The fourth and fifth fingers of each hand were examined on all subjects, as suggested by Hauptmann and Myerson (5), and final ratings were made by averaging the values obtained on all four fingers.

For photography of the capillaries, the same procedure was followed except that the microscope was equipped with a Micro-Ibso attachment supporting a Leica camera. An assistant manipulated the prism in the Micro-Ibso attachment and also the camera shutter when the examiner gave a signal that the capillaries were in focus.

Two projection lamps, one with a 500-watt bulb and the other with a 300-watt bulb, were

[1] For the assistance and cooperation without which this study would have been impossible, we wish to thank Dr. Nathan Kline, Mr. Ashton Tenney, and the other members of the Rockland State Hospital Research Facility; Dr. Robert Galambos, Dr. J. G. Beebe-Center and Dr. Miles Rogers of the Harvard University Psychological Laboratories; Dr. Stanley Garn of the Forsythe Dental Clinic; Dr. Milton Greenblatt of the Boston Psychopathic Hospital; Dr. Edward Budnitz of the Worcester City Hospital; Mrs. Eugene Trainor of the Friendly House, Worcester, Mass.; Mr. Horgan of the Worcester Fire Department; and Mr. Frederick Coe and Dr. Karl Benedict of Norton's Abrasives Industrial Plant. Finally, we acknowledge with gratitude a grant from the Research Committee of Wesleyan University to help cover the expense of reproducing the figures in this communication.

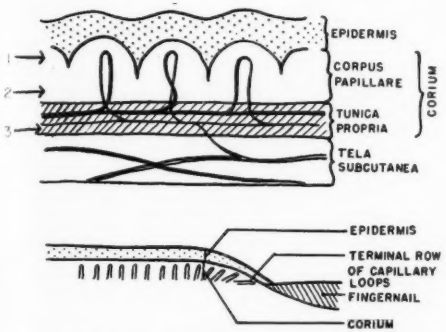


FIG. 1. Schematic diagrams of the skin. A (upper). Cross section, showing structure of skin capillaries. B (lower). Longitudinal section showing angle of capillary loops at different places on the finger.

found to provide adequate light for taking pictures at an exposure of 1/8 second, using the standard opening of the attachment. Green gelatin of a moderately dark shade was used as a filter for the light. Kodak Super-XX film was used and developed in Microdol. Prints were made on F-5 high contrast paper.

About 150 usable photographs were obtained on some 80 subjects. In the light of previous observation of some 300 subjects, it was felt that these photographs represented the range of variability of capillary patterns fairly well; they were therefore used as a basis for setting up rating scales. The scales illustrated in figures 2 and 3 resulted, the number of steps being different for the different scales since some of the variables presented a wider discriminable range than others. Half-steps were used in making the actual ratings of the nailbed.

THE SCALES

Representative photographs were chosen to define each step in each scale, and these served as a standard for all the ratings made in this study. The actual ratings, however, were made by direct observation rather than photography for photographs are necessarily limited as to breadth and depth of field.

Since the capillary picture is not always consistent over the whole nailbed, the entire field of a given nailbed was taken into account and an average or characteristic value decided upon. The end row of loops is often apparently different from loops observed proximally to it, so for standardization purposes all ratings of loops were made on the basis of this end row since the other rows are frequently oriented vertically to the skin surface and thus almost impossible to rate.

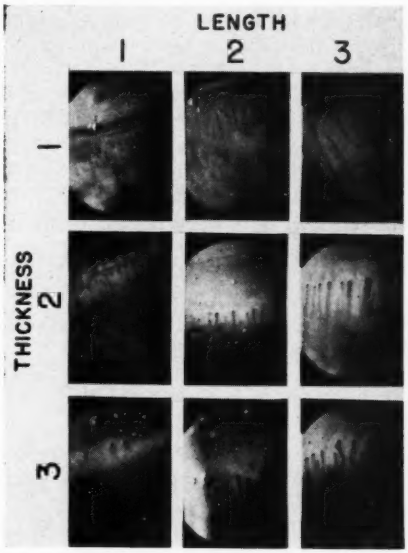


FIG. 2A. Scales of thickness and length of capillary loops. Photographs.

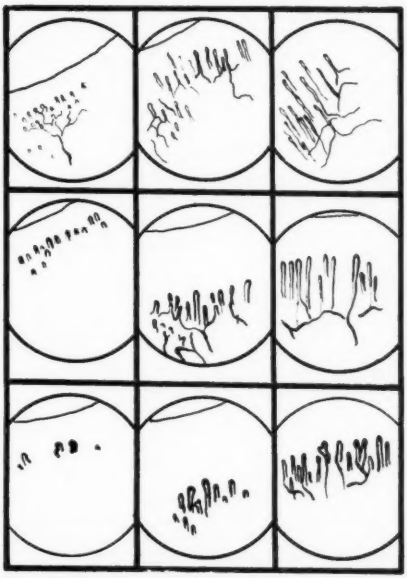


FIG. 2B. Scales of thickness and length of capillary loops. Tracings.

Thickness of loops (fig. 2). The thinnest loops are rated "1" on this scale; the thickest, "3". Thickness seems to be a measure of the width of the blood stream in the capillaries, which in turn is a function of the size of the vessel and the rate of flow of the blood (slow-flowing blood engorges the capillary and produces greater apparent width).

Table 2 shows that children have thinner vessels than adults, and this makes it seem likely that an increase in thickness is a normal developmental occurrence, at least to some extent. On the other hand, table 2 also shows that schizophrenics have thicker vessels than normal adults, and it seems unlikely that this is a matter of developmental growth. It is more probable that the schizophrenics were frequently exhibiting stasis of the blood and resulting engorgement of the capillaries.[2]

In summary, high thickness ratings may reflect increased anatomical size of the vessel (due conceivably to growth or to the body type), or they may measure the degree to which the vessels are engorged by a sluggish blood flow.

Length of loops (fig. 2). The shortest loops are rated "1"; the longest, "3". From figure 1 it is clear that several different factors may affect the apparent length of the capillary loop: first, the angle the capillary loop makes with the skin surface, a vertical loop appearing shorter than one lying horizontally; second, how far down into the skin the loop is visible (a function of the thickness of the skin); and third, how long the loop actually is from base to tip. When the plexus, or base of the loop, is visible, only the angle the loop makes with the skin surface is apt to interfere with determination of its actual length, and this factor is at a minimum at the end row where the loops tend to lie horizontally.

Since the plexus is more often visible in children than in adults (table 2), it is likely that the full length of the capillary loop is seen more often in the children. Thus it is likely that children do have shorter loops, on the average, than adults even though no such difference shows up in rating the length of the *visible* portion of the loop. Presumably length as well as thickness increases anatomically in the growing child.

In schizophrenics, as in children, the plexus is visible more often than it is in normal adults (table 2). Thus the finding that schizophrenics have longer loops than normals may be due entirely to the greater frequency with which the entire length of the loop is seen in schizophrenics.[3] In short, it seems likely that both the greater width and the greater length of capillary loops observed in schizophrenics are functions of variables other than actual anatomical size.

Plexus (fig. 3). Plexus is perhaps the only structural element listed here which cannot be adequately rated without searching the area for at least a few rows below the end row of loops. Its typical development is seen as a network of more or less horizontal vessels running along at the base of the loops. "0" indicates the absence of any visible plexus. "1" is used if only a few very short branches of plexus are seen, i.e., two loops joined together at the base by a short horizontal branch. "2" is used when a horizontal network is definitely visible, but localized in area, occurring only at one side of the finger, only close to the end row, or only for a limited number of rows (about two rows) at some area proximal to the end row. "3" refers to a clearly developed plexus, beginning at or nearly at the base of the end row and extending for some distance proximally. "4" is reserved for the most extreme degree of plexus, i.e., nailbeds showing not only the thin vessels but vessels much thicker than the capillary loops as well, or those where the plexus seems to "swamp" the loops so that it is hard to tell whether some of the loops are not actually just bumps in the plexus.

From fig. 1A it is clear that one must see through both the epidermis and the corpus papillare in order to see the plexus vessels. If either the epidermis or the corpus papillare is quite thick, the plexus will not be visible. Depending on the thickness of the layers, the examiner may be able to see to any of the different depths, such as those indicated by arrows 1, 2, and 3 in fig. 1A. If he sees to depth 1, the loops appear short, and no plexus is visible

[2] Bellak's *Dementia Praecox* (1, pp. 98–99) lists several references on slowed circulatory rate in schizophrenia, and actual observations of blood flow in our subjects bore out the stated interpretation.

[3] In the adult population studied, length of loop was positively correlated (.02 level of confidence) with plexus visibility, which supports this interpretation of the greater length ratings in schizophrenics.

(rated "0"); at depth 2 the loops seem longer (provided they are not oriented too vertically), and an occasional vessel from the plexus may rise up high enough to be visible (rated "1"). At level 3, the subpapillary plexus is clearly visible (rated "2" or "3", depending on whether or not the examiner can see to this depth over most of the nailbed). The visibility of heavy vessels lying below level 3 is one of the criteria for rating a plexus formation "4".

From this analysis, it looks as though the plexus rating is an integumentary measure rather than a circulatory one. This may well be, but the correlations between plexus formation and circulatory function (table 5 below) suggest that this is not the whole story. A thick epidermis may not be related to circulatory structure, but a thick corpus papillare probably is, indirectly, in that it is a mesodermal tissue which develops in close relationship to the capillary loops which project into it.

It should be emphasized that a few cases were seen where the subject almost certainly had abnormally thick epidermis (i.e., one patient continuously washed her hands). In these cases, the loops themselves appeared fuzzy in outline and hard to see. Generally, however, the loops were clear-cut, even when the plexus was not visible. In these cases it was assumed that the epidermis is seen through fairly easily, but that the plexus lies too deep in the mesodermal layer (corium) to be seen. The epidermal layer is quite transparent, particularly when coated with the cedar oil; thus when the plexus cannot be seen, the most probable explanation is a thick corium layer.

Presumably, then, a visible plexus indicates a thin corium layer, and as such is a rough indicator of mesodermal development in the circulatory system in general. A negative relationship found between mesomorphy and plexus (.01 level of significance) bears out this interpretation.

Twistedness of loops (fig. 3). Here it is important, as in estimating length, to work with the end row since the more proximal rows frequently appear more twisted, perhaps because their more vertical orientation foreshortens them considerably. The degree of twistedness is estimated primarily from the proportion of the loops that are twisted, and only secondarily from how twisted the individual loops appear. If only a rare loop is twisted, a rating of "0" is given. A rating of "1" means that several are twisted but many are fairly straight. When twistedness is obviously a characteristic of the loops, but there is still a fair number of straight ones, a rating of "2" is given, and "3" is reserved for the case where practically all of the loops are distinctly twisted.

The main indication is that twistedness is a developmental feature since children show less of it than adults (table 2). It also was found to show a slight positive correlation with blood pressure measurements.

Special cases (fig. 4). Various structures which are difficult to rate are sometimes encountered. One is the "bi-lobed" formation (occasionally even triple-lobed); another is a loop, typically shaped like a sprung hair pin, open at the lower end, often scarcely recognizable as a loop at all, which we have called an "unformed" loop since it was relatively more frequently found in children; a third is the so-called "stunted" capillary loop formation, which exhibits extremely short, thin loops, usually somewhat twisted, but so small that their shape is hard to determine exactly. The last three photographs in the row labeled "stunted" are good examples of this type of loop. The first photograph, however, is a very questionable case since no plexus is visible, making it impossible to be sure that the loops are actually short; they may only be oriented vertically to the skin surface. This is true also of those photographs we have labelled "indeterminate," in which the field is so ambiguous and the skin so relatively opaque that rating becomes very difficult.

These four types of capillary pictures were not included in the statistical work here as they were comparatively rare, but we have the impression that the bi-lobed, unformed, and stunted patterns are associated with plexus, and that relationships which hold for plexus would probably hold for these structures also, in direction if not in degree. The "indeterminate" pattern, on the contrary, is associated with absence of plexus.

Reliability of Scales

To determine the reliability of the scales, a group of subjects was rated twice at two weeks' interval by the same examiner, and another

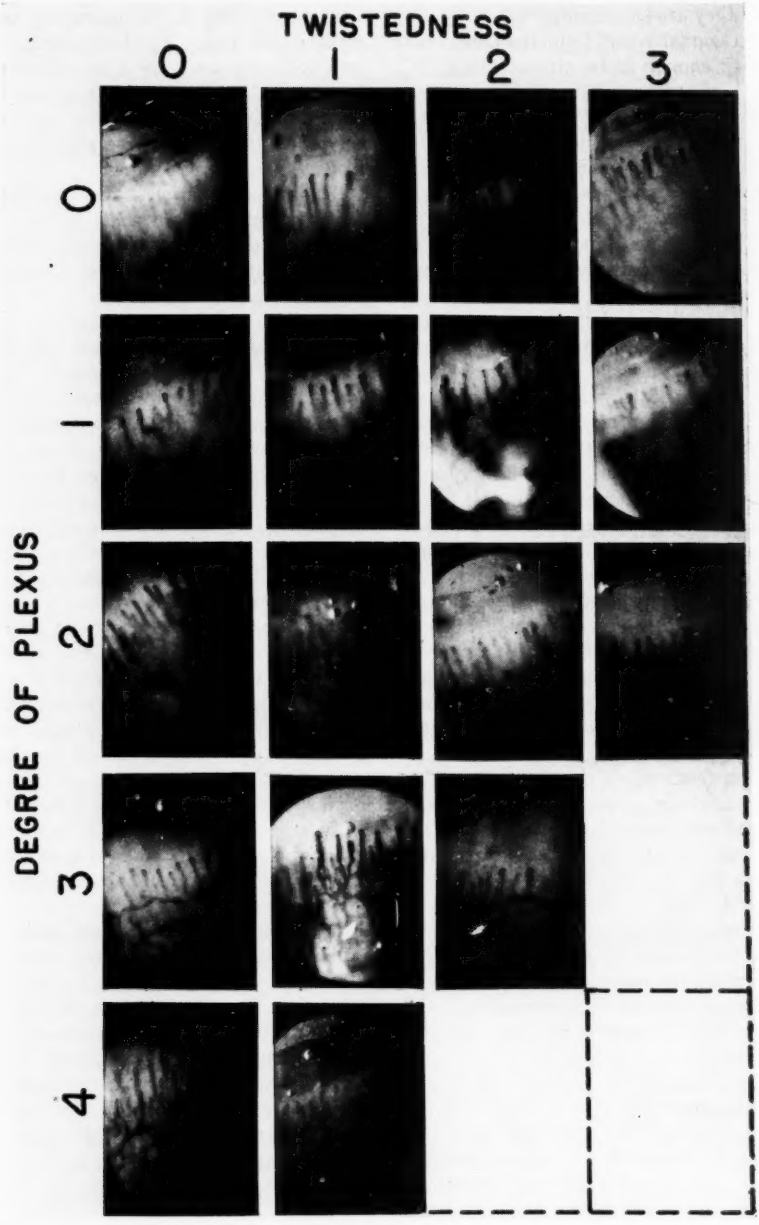


FIG. 3A. Scales of plexus visibility and capillary loop twistedness. Photographs

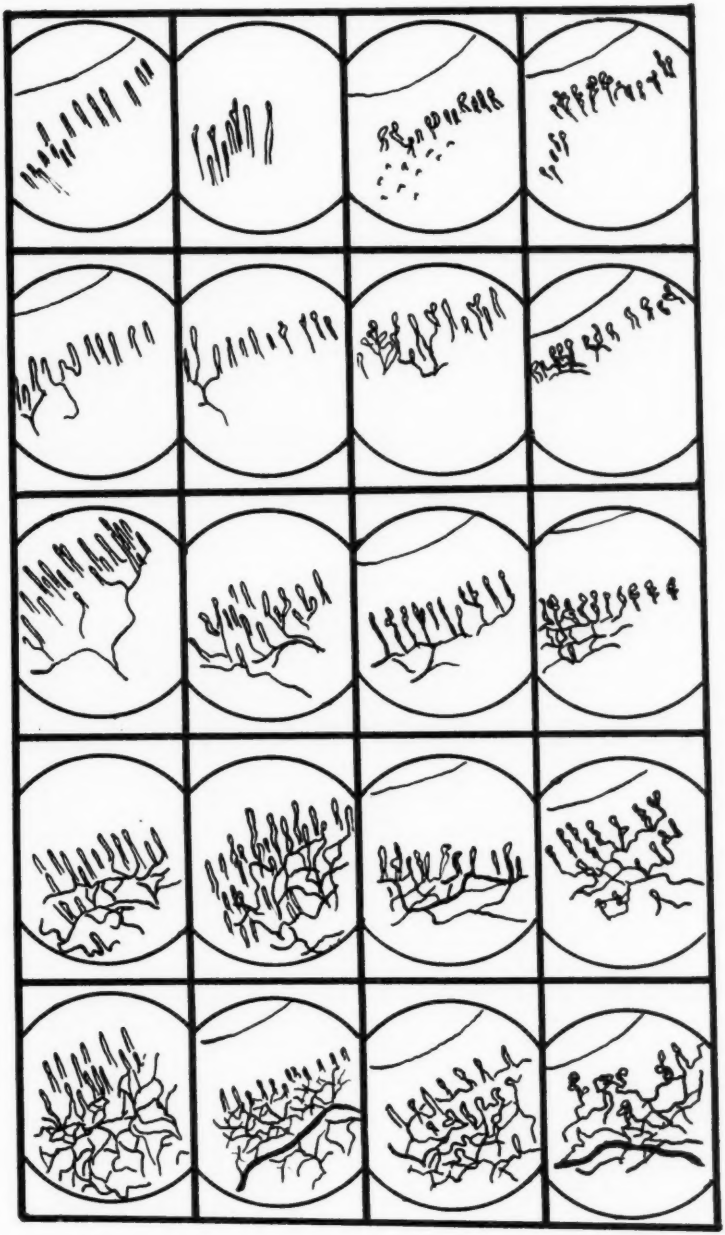


FIG. 3B. Scales of plexus visibility and capillary loop twistedness. Tracings

group was rated by two examiners, independently rating the capillaries within a few minutes of each other. For each of these two studies, table 1 shows (a) the percentage of second judgments that differed from first judgments by one-half a scale point or less; (b) the average difference between first and second ratings for each scale; (c) what percentage each average difference is of the total range of its scale; and (d) the rank-difference correlation (rho) between first and second ratings.

Some of the subjects used in this experiment had been examined in a preliminary investigation performed a year previously, making possible a third reliability study. Twist and plexus were both rated for these subjects at that time, but the scales used were slightly different. However, by ranking the scores for each indi-

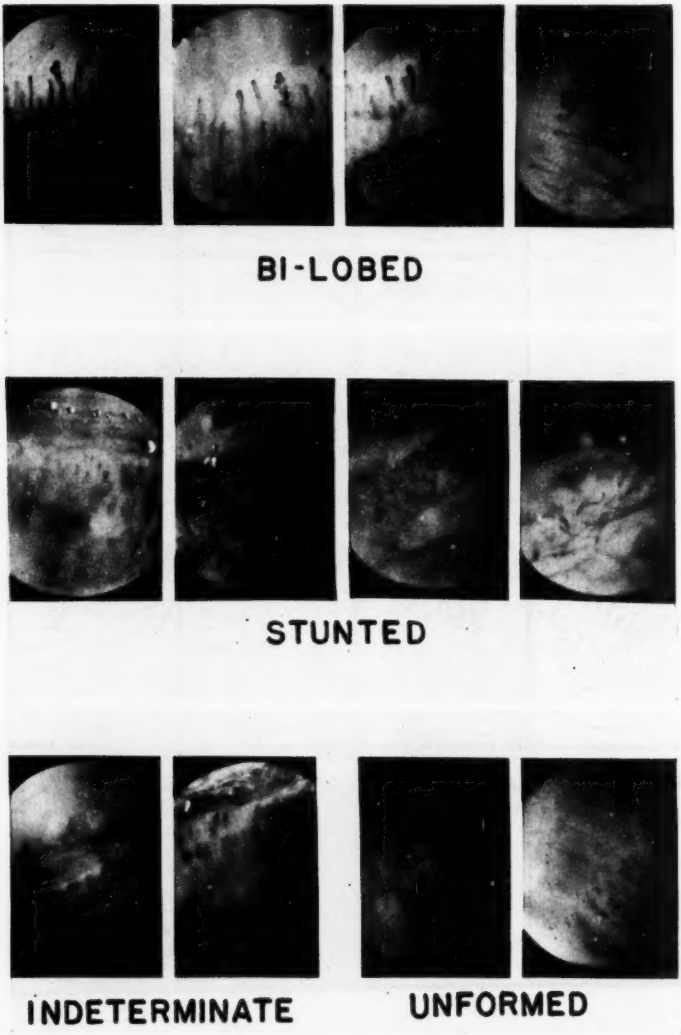


FIG. 4A. Special cases of capillary structure. Photographs

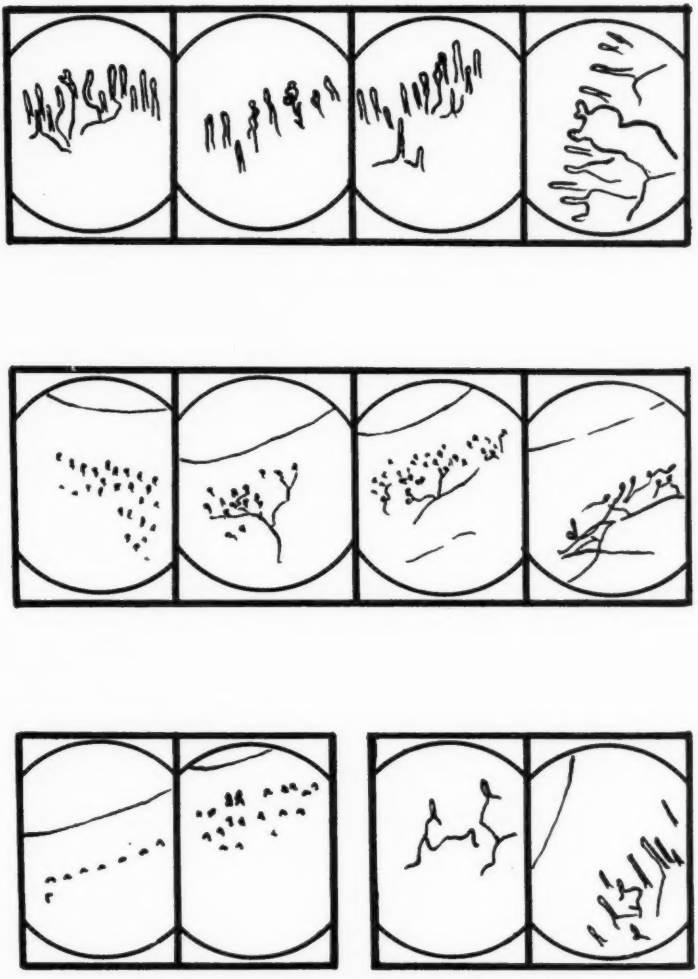


FIG. 4B. Special cases of capillary structure. Tracings

vidual in this group, a comparison was possible. The rank-difference correlations thus computed between ratings made by the same examiner a year apart were +.92 for plexus and +.70 for twistedness.

These results suggest that the scales are sufficiently reliable for a study of group differences.

SUBJECT-GROUP DIFFERENCES

Ratings were obtained on 58 psychotic men from the wards of the Worcester State Hospital; 31 psychotic women from the same source; 38 psychiatrically normal men, including firemen, laborers, hospital staff, students, and a few patients from a medical clinic; 25 psychiatrically normal women, including nurses, office workers, and students; and 41 children, ranging in age from 2 months to 14 years.

Table 2 shows the differences obtained between children, normal adults, and schizophrenics on the several capillary variables. In each case, the percentage of subjects falling

TABLE 1.—RELIABILITY OF THE CAPILLARY RATINGS

Scale	Per cent within ½ Point	Average Difference	Per cent of Range	rho
A. Comparison of ratings made by the same examiner at two weeks' interval (N = 19)				
Thickness	95	.21	10.5	+.84
Length	84	.27	13.5	+.78
Plexus	100	.20	5.0	+.93
Twistedness	89	.31	10.0	+.84
B. Comparison of ratings made by different examiners (N = 23)				
Thickness	90	.18	9.0	+.79
Length	100	.13	6.5	+.95
Plexus	100	.17	4.0	+.83
Twistedness	90	.28	9.0	+.85

TABLE 2.—GROUP DIFFERENCES IN CAPILLARY STRUCTURE. SEE TEXT FOR EXPLANATION

N		Rating		Chi Square	P
		Less than 2	2 or more		
		%	%		
	Thickness			9.6	.01
41	Children	71	29		
63	Normals	60	40		
83	Schizophrenics	42	58		
	Length			9.4	.01
	Children	62	38		
	Normals	56	44		
	Schizophrenics	30	70		
		Less than 1	1 or more		
		%	%		
	Plexus			14.9	.001
	Normals	79	21		
	Schizophrenics	49	51		
	Children	45	55		
		Less than 1.5	1.5 or more		
		%	%		
	Twistedness			6.4	.05
	Children	67	33		
	Schizophrenics	50	50		
	Normals	41	59		

above and below a certain dividing point on the rating scale indicated is recorded for each group, together with the probability (P), by Chi square, of the obtained differences occurring by chance. (Six of the psychotic patients could not be included in table 2 since they showed such deviant patterns as the "stunted" capillaries pictured in fig. 4). Male and female subjects are lumped together here for economy of presentation; the same trends obtain for each sex taken separately.[4]

The differences that most interest us here are those between the normal and schizophrenic groups. The schizophrenics show thicker, longer capillary loops and more visible plexus than do the normals. This is consistent with a theory of hypoplastic circulatory structure in the schizophrenic group, the larger loops reflecting stasis of the blood with consequent engorgement of the capillaries, while the greater visibility of the plexus indicates a poorer development of the circulatory mesoderm.

A possible explanation of these differences would attribute them to the different occupations of the two groups and to the long hospitalization of the patients. At first glance this seems a very profitable hypothesis since, of the 38 members of the normal male group, 17 were either laborers or firemen, and 88 per cent of these had plexus ratings of less than "1" as opposed to only 67 per cent of the rest of the control group, consisting of hospital workers, students, and a few patients from a medical clinic.

On further thought, however, it becomes clear that the firemen spent a great deal of their time simply sitting in the fire station waiting for an alarm; the laborers worked in an abrasives plant. The occupation of the latter group would be expected to produce thick skin on the hands to a larger extent than the former, and yet 92 per cent of the firemen had plexus ratings under "1", as compared to only 80 per cent of the laborers.

The fact that many of the patients tested worked daily in the laundry or on the hospital farm is further evidence that occupation cannot be an adequate explanation of the differences found; and one would also, on this hypothesis, expect to find a relation between duration of hospitalization and the capillary variables; none of the variables, however, was found to be related to duration of hospitalization.

The hypothesis of constitutional differences

[4] A more detailed workup of the data is available elsewhere (13).

to account for the difference in structure was better supported. We used somatotype (12) rating to indicate morphological differences, presumably with a constitutional basis. The somatotypes were obtained from Dr. William Sheldon, who performed the ratings on standard medical photographs taken of the patients and normal males. Unfortunately, no somatotypes were available on the normal females.

Here, as in occupation, the hospital workers, students, etc. differed from the laborars and firemen. Of the 17 men in the latter group, 9 (53 per cent) had mesomorphy ratings of 5 or more, while only 2 of the 9 somatotyped members of the former group had such ratings (29 per cent). All those in either group who showed a plexus rating of more than 1 were below 5 in mesomorphy.

When somatotype ratings were compared with capillary ratings, both the normal males and the schizophrenic females showed a significant negative relationship between plexus and mesomorphy rating. The same trend was present in the schizophrenic males, but not significantly, probably due to a relatively curtailed range of the mesomorphy variable for this group. The critical point on the mesomorphy scale, so far as the capillary variables are concerned, was at a rating of "5" for the males, "4" for the females. Visible plexus and engorged vessels seemed to occur only very rarely when mesomorphy was this high or higher.

Since normal females were not somatotyped, the relationship between capillary structure and morphology was investigated only for the male population. The normal males showed somewhat more mesomorphy and less endomorphy than the schizophrenic males (.06 level of significance). In order to more nearly equate the two groups, therefore, all subjects in *either* group who had mesomorphy ratings of "5" or more, or endomorphy ratings below "2", or both, were excluded; and the curtailed groups thus obtained were compared on the capillary variables.

As can be seen in table 3, the normal and schizophrenic groups no longer showed appreciable differences in plexus ratings when they were thus adjusted for somatotypic differences. Differences in length and thickness were also curtailed, but less markedly. No significant differences between schizophrenic and normal

TABLE 3.—Plexus Rating as a Function of Diagnostic Group

	Plexus Rating		Chi Square	P
	Less than 1	More than 1		
A. All males on whom somatotypes were available				
	%	%		
Normals, N = 26	81	19	6.7	.01
Schizophrenics, N = 50	50	50		
B. Males rated both less than 5 in mesomorphy and more than 2 in endomorphy				
Normals, N = 12	58	42	0.3	—
Schizophrenics, N = 39	46	54		

subjects remained after the groups were adjusted for somatotype, but this is not conclusive evidence that differences were eradicated; the N was sharply decreased in the process of equating the two groups, and the significance values would have decreased even if the proportions of cases had remained the same. However, the fact that the distribution of capillary structures is so similar for the normal and schizophrenic groups, once they are equated for somatotype, is presumptive evidence that differences in capillary structure originally found between the two groups were probably due in large part to the fact that 54 per cent of the normal group had mesomorphy ratings of "5" or more and/or endomorphy ratings below "2", while only 22 per cent of the schizophrenic group could be so classified.

Diagnostic-Group Differences

Table 4 shows the differences between diagnostic groups. Males and females are grouped together to form these diagnostic categories since the groups would have been rather small otherwise. No tendency for a sex difference to obscure or reinforce a trend was noted, however.

Of those on whom diagnoses were available[5] 28 were called paranoid schizophrenia, and 28

[5] Several of the patients were only diagnosed dementia praecox, with no further specification. They, as well as manic-depressives, mental defectives, etc., were not included in this analysis as we had too few in our samples to give us adequate numbers of cases in each group.

TABLE 4.—Capillary Ratings as a Function of Diagnostic Group

N		Rating		Chi Square	P
		Less than 2	2 or more		
		%	%		
	Thickness			3.4	.20
63	Normals	60	40		
28	Paranoids	38	62		
28	CHS Schizophrenics	37	63		
	Length			20.6	.001
	Normals	56	44		
	Paranoids	36	64		
	CHS Schizophrenics	21	79		
		Less than 1	1 or more		
		%	%		
	Plexus			13.6	.001
	Normals	79	21		
	Paranoids	52	48		
	CHS Schizophrenics	37	63		
		Less than 1.5	1.5 or more		
		%	%		
	Twistedness			4.9	.10
	Paranoids	38	62		
	Normals	41	59		
	CHS Schizophrenics	66	34		

either catatonic, hebephrenic, or simple schizophrenia. These last three diagnoses were lumped into one group partly because there were so few cases in each diagnostic category, and partly because there are some indications that these categories form a group which is fairly homogeneous on many physiological measures and different from the paranoids. The diagnoses were not made known to the examiner until after capillary ratings had been made.

Table 4 shows that the paranoid schizophrenics fell between the CHS (catatonic, hebephrenic, and simple) schizophrenics and the normals for each of the capillary scales except twistedness. The paranoid group seems to have shorter loops and more twisted capillaries, on the average, than the CHS group, and significantly less plexus. Although this makes the paranoid group more similar to the normals than to the schizophrenic group as a whole, they, in turn, still have significantly longer loops than normals, and more plexus.

Somatotype showed no significant relation with type of schizophrenia for this sample, nor could the difference between the CHS schizophrenics and the paranoid schizophrenics be explained by differences in somatotype, as the differences between normals and schizophrenics seemed to be. This is not surprising since mesomorphy ratings below "5" showed little relation to capillary structure and most of the schizophrenics fell into this lower mesomorphy class. It is interesting that, for the particular function of separating the CHS schizophrenics from the paranoids, the capillary structure was more efficient than the somatotype, at least for this sample.

Since diagnostic categories are notoriously unreliable in psychosis, another method of distinguishing between types of psychosis was investigated. The onset age was estimated for each patient from his case history. In order to keep the estimate objective, the date of first hospitalization was considered to be the date of onset. Obviously this date is rarely accurate, but it was hoped that the error involved would be random except for the constant error involved due to the fact that hospitalization always follows, rather than precedes, onset.

It was found that the date of first hospitalization of the schizophrenics bore a negative relation, at the .01 level, with plexus visibility (the more plexus, the earlier the hospitalization). Onset age seems to be the important factor here since neither duration of illness nor age of subject (over the adult age range) was significantly related to plexus development.

None of the somatotype components showed a significant relation to onset age for this experimental sample.

These differences found between paranoid schizophrenics, on the one hand, and catatonic, simple, and hebephrenic schizophrenics, on the other, are in accord with the reports in the literature. The greater degree of plexus visibility and the lower degree of twistedness found in the non-paranoid group corroborate the findings of Hauptmann and Myerson (5) although our paranoids did not show *no* plexus for this sample as they did in the Hauptmann-Myerson sample. In general, the fact that, for

all capillary measures, the paranoid schizophrenics were more similar to the normal population than were the non-paranoid schizophrenics, has precedent in earlier findings. Not infrequently the literature on physiological differences in psychosis will report that the paranoid group (along, usually, with the manic-depressive group) differs from the other schizophrenic groups, and that this difference is in the direction of the value found for the normals, sometimes even exceeding the normal-group value. Lemere's data on alpha rhythm indicate this (8), for example,[6] and Shattock's data on testicular tubular atrophy (11) differentiate the paranoids from the hebephrenics and simple schizophrenics, although not from the catatonics. The body type literature also suggests this trend (2, 7, 10); and most pertinent here, perhaps, is Lewis' report that heart size differentiates the paranoids from the non-paranoid schizophrenics (9).

Lewis reports that the small hearts which he frequently found in non-paranoid schizophrenics are hypoplastic, not atrophied, and, in fact, frequently show a failure to increase in size even in the presence of a lesion which would ordinarily lead to compensatory hypertrophy. He also reported that he has demonstrated small visceral capillaries in cases with small hearts.

Since Lewis found small hearts in the same diagnostic group that in Hauptmann and Myerson's and our own study showed greater than average plexus visibility, it seems reasonable that the two features may be related. Our view that visibility of plexus may indicate a sub-normal mesodermal development provides a link between the two findings since hypoplastic heart tissue would also be an expression of low mesodermal development. The stasis and engorgement of the capillaries hypothesized in many of the schizophrenics are also consistent with a hypoplastic circulatory system since the vessel walls in such a system are reported by Lewis to be quite elastic and distensible, and the pumping force of the heart might be assumed to be less adequate.

[6] Lemere's data (8) show less alpha rhythm in all subtypes of schizophrenia, but particularly markedly in non-paranoid schizophrenia. This finding becomes especially illuminating when considered together with Darrow and Graf's report (3) that a drop in blood flow in the brain decreases the alpha rhythm.

If the capillary structure found in schizophrenia does suggest a hypoplastic circulatory structure, an interesting theory arises as to the correlation found between plexus and age at first hospitalization. Schizophrenia, especially of the non-paranoid types, is generally considered to have its onset at the time of, or shortly after, adolescence. Adolescence is also a time when the circulatory structure undergoes normal growth or hypertrophy. If, as Lewis suggests, some circulatory systems seem relatively incapable of hypertrophy, even in the face of conditions requiring such growth, the people possessing them might be considered to enter into a critical period as they undergo adolescence. The demands on the circulatory system are increased, and the system's ability to meet these increased demands is limited.

It is conceivable that the presence of visible plexus is a gross indicator of some such handicap. Such a handicap would not, of course, necessarily result in any breakdown in the system; a large number of other factors, environmental and constitutional, would inevitably contribute to adjustment or maladjustment of the system.

In the foregoing discussion, there have been indications that the development of mesodermal structures may be minimal in many schizophrenics, particularly the simple, hebephrenic, and catatonic patients. Primarily this has been manifest in the circulatory system, and perhaps subnormal development of this system is more important than low mesodermal development in general since the capillary measures employed differentiated among the schizophrenics and normals, and the various types of schizophrenia, better than the mesomorphy rating (the latter presumably being more a measure of mesodermal development *in general*).

It is, for example, easy to hypothesize crudely that an inadequate circulatory system could contribute to schizophrenic breakdown by some such mechanism as reduced oxygen supply to the brain in times of stress. However, it should be borne in mind as at least likely that the circulatory differences here indicated are not at all directly involved in the etiology of schozophrenia, but are more or less coexistent with other more important differences.

Functional Studies

There are many reports in the literature of measures of bodily function which differentiate significantly between schizophrenics and normals. One important control that is rarely observed in these studies is the control on morphology. Schizophrenics may be functioning differently than normals, but at the same time they may, if there are differences in morphology between the two groups, be functioning *normally for their particular structure.*

Hoskins mentions that blood pressure and pulse rate in schozophrenics are generally low (6), and Freeman *et al.* (4) show this in their data. Hoskins also reports a lowered basal metabolic rate in schizophrenics.

In this study, basal blood-pressure and respiration-rate measures were available on 14 subjects, and blood-pressure measures available on 11 more. BMR measures were also taken on a group of 17 of the subjects. All these data were obtained by people unacquainted with the capillary ratings made on the subjects. Table 5 shows the relationships obtained. Only the plexus variable is tabulated; the other capillary scales did show some relationships, but not such significant ones as did the plexus scale. From table 5 it will be seen that the high plexus characteristic of the schizophrenic group is found in conjunction with low blood pressure and BMR values. Thus it is not impossible that the relatively low blood pressure and BMR values reported for the schizophrenics are, in fact, closely related to the different circulatory structure of the schizophrenics or to some other related morphological characteristic.

This does not imply that the particular functioning level of the schizophrenic is unimportant in the etiology of the disease; but it does shift the emphasis and indicates a different road of attack on the basic problem. Rather than saying that the schizophrenic is functioning abnormally in certain respects, it may sometimes be more profitable to say that he is functioning normally for his structure, but that his structure is not equipped to handle the particular situation (physiological or social) in which he finds himself.

TABLE 5.—Relation of Certain Functional Measures to Plexus Rating

Measure	N	Direction of Relation	P
Systolic blood pressure	25	Negative	.002
Pulse pressure	25	Negative	.002
Basal metabolic rate	17	Negative	.02
Respiration rate	14	Positive	.002

Summary

1. Four scales for rating capillary structure have been presented (scales of thickness, length, and twistedness of loops, and degree of sub-papillary plexus visible), with photographed standards.

2. As rated by these scales, schizophrenics showed longer, thicker loops than normal adults, and a greater degree of plexus. It is suggested that the thicker loops of the schizophrenics are due to stasis of the blood and engorgement of the capillaries, while the greater length and plexus visibility are attributed to an underdevelopment of the mesodermal layer of the skin in which the capillaries lie.

3. Paranoid schizophrenics showed capillary pictures more similar to the normal than did a combined group of catatonic, hebephrenic, and simple schizophrenics. The two schizophrenic groups differed significantly from each other as well as from the normal on the different capillary measures, particularly on plexus ratings.

4. Age of first hospitalization was negatively related to degree of plexus in schizophrenics. The possibility that this is related to failure of the usual hypertrophy of the circulatory system during adolescence is discussed. Neither age at examination nor duration of illness was significantly related to plexus in the adult groups.

5. A negative relation was demonstrated between mesomorphy rating and degree of plexus, supporting the view that low mesomorphy and high plexus are both manifestations of poor mesodermal development.

6. Schizophrenic males were shown to have somewhat more endomorphy and less mesomorphy than normal males, and correction for this difference essentially removed the differences in capillary structure found between the two groups. No significant relationships could be demonstrated between somatotype and

diagnosis of the patients or date of first hospitalization, however.

7. Relationships between capillary structure, on the one hand, and blood pressure, BMR, and respiration rate, on the other, were presented, and the possibility mentioned that differences found between schizophrenics and normals on these functions might be explainable on the basis of structural differences between the two groups.

BIBLIOGRAPHY

1. Bellak, L.: *Dementia Praecox.* New York: Grune & Stratton, 1948.
2. Betz, B. J.: Somatology of the schizophrenic patient. *Human Biol., 14:* 192–234, 1942.
3. Darrow, C. W., and Graf, C. G.: Relation of the EEG to photometrically observed vasomotor changes in the brain. *J. Neurophysiol., 8:* 449–462, 1945.
4. Freeman, H., Looney, J. M., Hoskins, R. G., and Dyer, C. G.: Results of insulin and epinephrine tolerance tests in schizophrenic patients and in normal subjects. *Arch. Neurol. & Psychiat., 49:* 195–203, 1943.
5. Hauptmann, A., and Myerson, A.: Studies of finger capillaries in schizophrenia and manic-depressive psychoses. *J. Nerv. & Ment. Dis., 108:* 91–108, 1948.
6. Hoskins, R. G.: *The Biology of Schizophrenia.* New York: Norton, 1946.
7. Kretschmer, E.: *Physique and Character.* London: Harcourt, Brace, 1925.
8. Lemere, F.: Electroencephalography. *Psychiat. Quart., 13:* 5–15, 1939.
9. Lewis, N. D. C.: Constitutional factors in dementia praecox with particular attention to the circulatory system and to some of the endocrine glands. Washington, D. C. *Nerv. & Ment. Dis. Monograph, 35,* 1926.
10. Rees, W. L.: Physical constitution, neurosis, and psychosis. *Proc. Roy. Soc. Med., 37:* 635–638, 1944.
11. Shattock, F. Mackenzie: The somatic manifestations of schizophrenia. A clinical study of their significance. *J. Ment. Sci., 96:* 32–142, 1950.
12. Sheldon, W. H., Stevens, S. S., and Tucker, W. B.: *Varieties of Human Physique.* New York: Harper, 1940.
13. Wertheimer, N.: The relation of capillary structure at the nailfold to certain psychological and morphological variables. Unpublished Ph.D. thesis, Harvard University, Cambridge, Mass., 1954.

WESLEYAN UNIVERSITY
MIDDLETOWN, CONNECTICUT

FOCAL ABNORMALITIES OF THE ELECTROENCEPHALOGRAM IN JUVENILES WITH BEHAVIOR DISORDER

ELIZABETH JONES, M.D., B. K. BAGCHI, PH.D., AND R. W. WAGGONER, M.D.

The value of EEG in the detection of cerebral disorders in juvenile delinquents is gaining increasing recognition among psychiatrists and legal agencies. We have reviewed the EEG records of 390 children and adolescents (2 to 18 years of age) with behavior disorders treated at the University Hospital from 1941 to 1954 (December). In this group of 390 children with behavior disorders there were 47, or 12 per cent, with some type of *focal* EEG abnormality. It is the analysis of this group of forty-seven cases which is to be discussed here. Focal EEG abnormality is considered here to refer to abnormality either in one or more areas of one hemisphere or in homologous areas of both hemispheres.

According to Kennard and Willner (9), Jasper, Solomon, and Bradley (8), Strauss, Rahm, and Barrera (17), and others (3, 2, 10, 5, 16) there is an incidence of 60 to 80 per cent of abnormal EEG's in children and adolescents with various disorders of behavior as compared with 10 to 16 per cent of abnormality in the same age groups of the normal population (9, 10, 5, 16, 7). The EEG abnormalities in cases of behavior disorder have mostly been described as diffuse in type and designated as "immature," "dysrhythmic" or "epileptoid," but there are six reports (8, 17, 12, 6, 19, 15) in which localized EEG abnormalities have been noted.

In our group of 47 cases, 28, or 60 per cent, had past history of some contributory or associated condition: (a) 15 had a history of birth injury or premature birth, skull fracture, severe closed head injury, or "encephalitis"; (b) 6 patients had convulsive seizures and some other contributory factor; (c) 2 were mentally deficient, had convulsions, and had in addition at least one of the above-named contributory factors; (d) 3 children had convulsive seizures only; and (e) 2 were mentally deficient. In 19 cases, or 40 per cent, there was no significant previous organic history (table 1).

From these 47 cases, 5 were selected to illustrate various focal abnormalities in the EEG of types similar to those which occur in epileptics.

Case 1. J. G., a 16-year-old boy, was referred by the juvenile court after he had impulsively and without provocation attacked a teen-age girl with a hammer. He stated that "sexual feelings" motivated the attack. There was no history of any contributory abnormal condition, convulsive episodes or significant past illness. His I.Q. was 112 on the Wechsler-Bellevue test. He was classified as psychoneurotic with uninhibited impulses. The EEG demonstrated in the right temporal lobe a focus of high voltage, sharp, single alpha spikes (fig. 1-a), alpha spikes with 3.5–4 p.s. components (fig. 1-b), and sharp diphasic spikes and double alpha bursts (fig. 1-c). During hyperventilation there occurred generalized high voltage 6–7 p.s. theta waves. On the basis of the findings in the EEG, the clinical picture was interpreted as convulsive disorder of psychomotor type with localization in the right temporal lobe. Psychotherapy was given while he was in the hospital. He was discharged 3½ months after admission, and his condition was considered improved. He received no anticonvulsant medication.

Case 2. T. F., a 12-year-old boy, had periods of violent anger during which he repeatedly destroyed property, without provocation threw a chair at a playmate, and attempted to choke another boy. These outbursts were increasing in frequency and severity. There was no history of injury or significant illness. He had had no convulsions. His I.Q. was 131 on the Wechsler scale for children. On neurological examination an almost constant myoclonic activity of the hands, face and neck was noted. The EEG showed a shifting temporal focus of grouped high voltage multiple alpha spikes and masked 3 p.s. waves (fig. 2). A prompt and abnormal effect was produced with hyperventilation.

Episodes of rage, myoclonic activity and the

TABLE 1.—E.E.G. IN BEHAVIOR DISORDERS
47 out of 390 cases showed focal abnormality

A. Behavior Disorder with Organic Factors		28	60%
(a) Birth injury, prematurity, head trauma, skull fracture or encephalitis	15		
(b) Convulsive disorder and one or more from (a)	6		
(c) Mental deficiency, convulsive disorder and one or more from (a)	2		
(d) Convulsive disorder without (a)	3		
(e) Mental deficiency	2		
B. Behavior Disorder without Organic Factors		19	40%
Total		47	

findings in the EEG suggested what has been described as a "centrencephalic" abnormality or dysfunction of other deep structures. He was treated with phenobarbital, Dilantin, Phenurone and later with Serapil. Psychotherapy was also used. At the time of this writing, 4 months after the initiation of serpasil therapy, there was some improvement in behavior.

Case 3. L. McN., an 8½-year-old boy, was examined in the out-patient department of the University Hospital because of difficulty at home and in school. He had history of pneumonia and grand mal seizures as an infant and concussion of the brain at 4½ years without sequellae. His mother stated that he did not "seem to catch on," was restless and difficult to correct and had frequent nightmares. His teachers described him as "nervous, mentally disturbed, and confused." His memory was so poor that in spite of tutoring he would forget what he had learned the previous day. He was unable to find his way home or about the school although he had been travelling along the same route for months. There were episodes of peculiar behavior at school during which he would suddenly jump up on a desk, yell or yawn loudly. He apparently was not aware of these spells. He also exhibited peculiar rocking and various purposeless, rhythmic movements. At other times he appeared dazed or in a dreamy state but had no attacks of typical petit mal. He suffered with nausea, vomiting, abdominal pain and pallor which were not associated with confusion. His I.Q. was 96 on the Stanford-Binet test. The neurological examination revealed no significant abnormality. The EEG showed abnormal wave formations consisting of abortive 3 p.s. spike-waves or triangular waves with a slow positive decaying phase and single or serial genuine

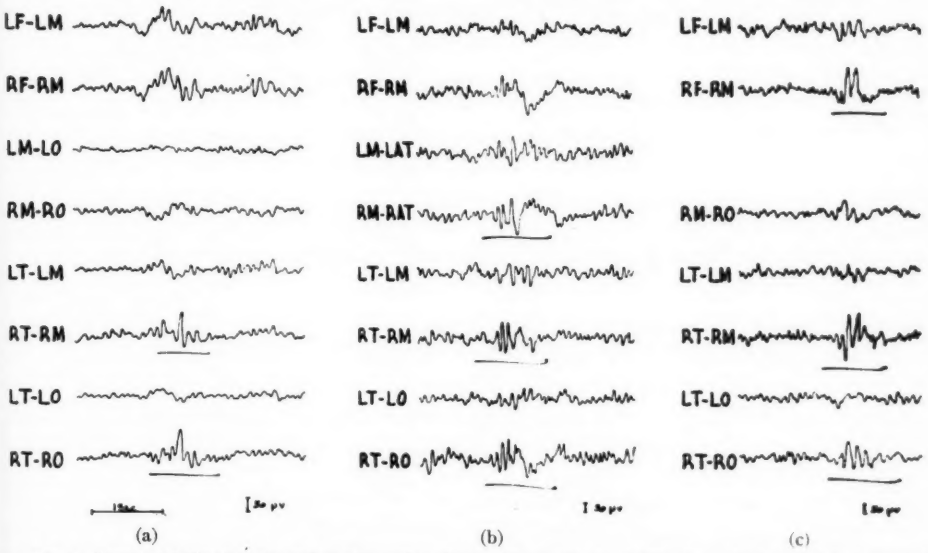


FIG. 1. Case 1. (a) Single alpha spikes of negative phase with amplitude of 150 microvolts in the right temporal lobe as shown by the underlined areas; (b) single and double alpha bursts with associated single 3.5 to 4 p.s. single waves; (c) double alpha bursts and sharp, diphasic waves in the right temporo-motor-occipital lead combinations.

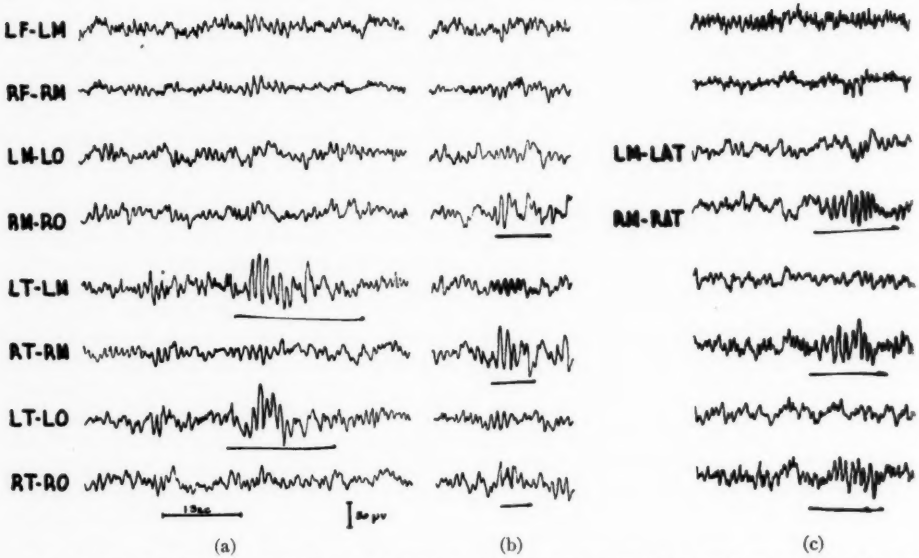


FIG. 2. Case 2. Shifting temporal focus of 4–5 p.s. serial waves with grouped high voltage multiple alpha and masked 3 p.s. waves; (a) in the left temporal region; (b) shift to the right temporal; (c) voltage emphasis in right anterior- and mid-temporal regions.

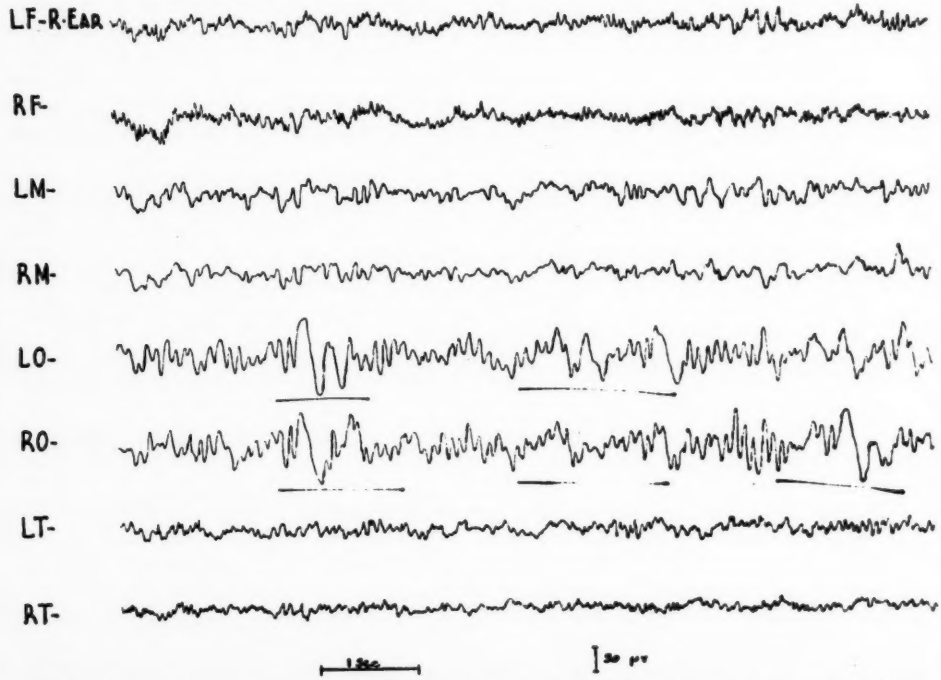


FIG. 3. Case 3. The underlined areas demonstrate abortive 3 p.s. spike and waves which occur as single complexes or short serial formations bilaterally in the occipital regions. In many instances the slow wave of the complex is positive in the decaying phase.

spike-waves in both occipital areas (fig. 3). He was treated with phenobarbital; a follow-up 8 months later revealed that he had marked improvement in behavior and had no spells or convulsive seizures.

Case 4. R. D., a 16-year-old boy, had periods of aggressive behavior, psychomotor and grand mal seizures and sexual aberrations (masturbation and sexual advances to both boys and girls) for two years; he was also delinquent and truant. He had had frequent encounters with the police and was referred here by a probate judge. There were no contributory organic factors. His I.Q. was 96 on the Wechsler-Bellevue scale. The initial EEG at the age of 14 showed a definite focus of high voltage 1.5–2 p.s. slow waves in the right temporal region which at times assumed the appearance of abortive spike and wave formations (fig. 4). Generalized high voltage discharges were produced by hyperventilation. He was treated with various anticonvulsant drugs including a brief therapeutic trial of Phenurone and later with Dilantin. A repeat EEG 3 years after the initial record showed a well-organized background of alpha activity and disappearance of the slow wave focus (fig. 5). He was gainfully employed, and there was no outward evidence of abnormal behavior. During the next 2 years he discontinued medication and subsequently again developed behavior difficulties.

Case 5. K. M. was a 9½-year-old boy who according to the statement of his mother had always been a "nervous child," was enuretic, restless, had rage reactions, was destructive and a firesetter. He ran away from home, had difficulty in school and was subject to nightmares. He liked to dress in feminine clothes. There was history of severe and recurrent ear infections two years previously, after which his behavior difficulties were aggravated and psychomotor spells occurred. A pneumoencephalogram showed subarachnoid air over the right cerebral hemisphere, but no other abnormality, and there were no definite signs of arachnoiditis. His I.Q. was 126 on the Stanford-Binet test. In the EEG there was an almost continuous focus of high voltage 2–3 p.s. delta waves in the right posterior quadrant, triangular or irregularly rounded in type (figs.

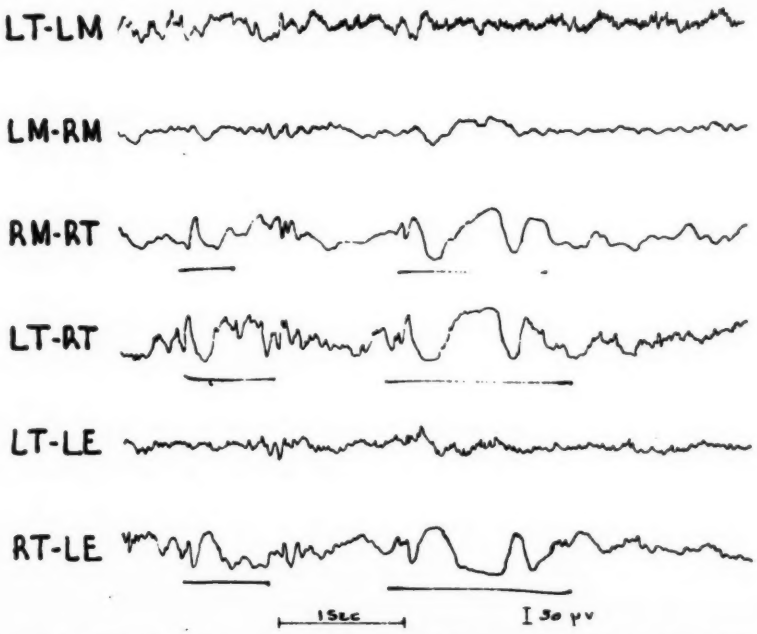


FIG. 4. Case 4. The initial EEG shows 1.5 to 2 p.s. rounded, high voltage waves in the right motor-temporal area (strip 3), with the left temporal-right temporal lead combinations (strip 4), and in right temporal lead with left ear as ground lead (strip 6).

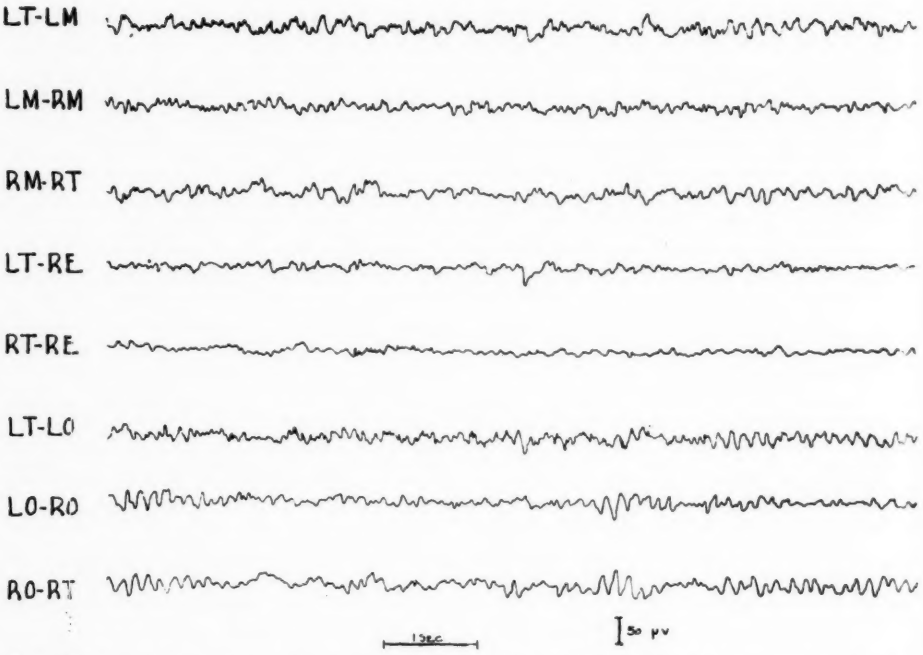


FIG. 5. Case 4. Second EEG taken 3 years after the initial one. The background pattern consists of medium voltage 9 to 9.5 p.s. alpha waves. There are some borderline features consisting of scattered theta waves; there are no delta waves.

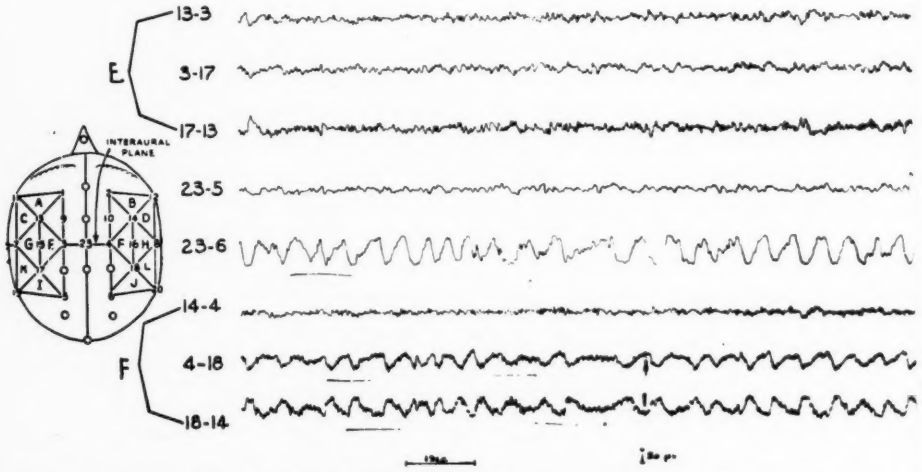


FIG. 6. Case 5. The upper three strips represent the left interauralmedial triangle "E"; the lower three strips (triangle "F"), the homologous area on the right; 4th and 5th strips are the recordings from the left and right parieto-occipital leads, respectively, with vertex lead as reference. There are almost continuous high voltage 2-3 p.s. focal delta waves in the underlined right parieto-occipital regions.

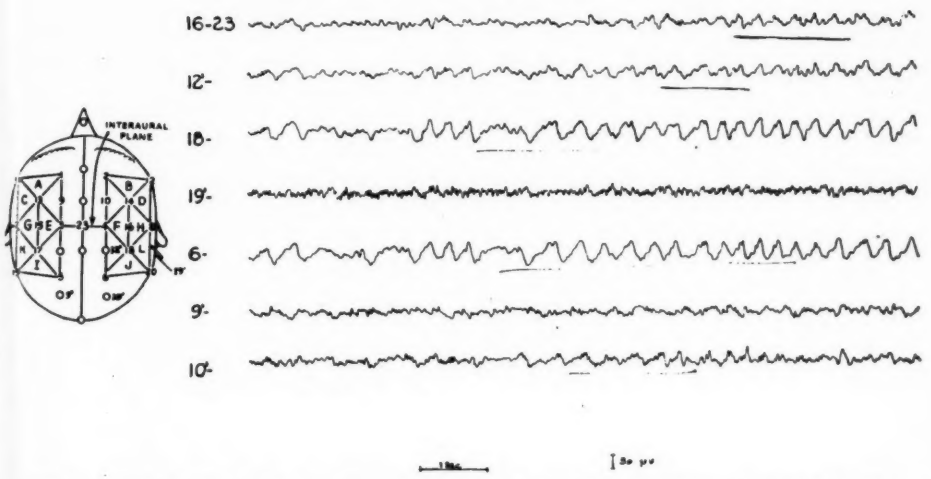


FIG. 7. Case 5. Another epoch of the EEG shown in fig. 6. Comparison of different areas on the right with vertex as common reference. Highest voltage of focal waves occurs in the lateral parietal and parieto-occipital regions.

6 and 7). The areas most involved were the lateral parietal and parieto-occipital regions. The patient was treated with Dilantin, phenobarbital and Phenurone. He continued this medication and had had no behavior difficulties at the time of a follow-up 4 years later. A repeat EEG could not be obtained.

COMMENT

In the group of 47 cases with focal abnormality of the EEG, there were 28 patients with a history of either trauma of the head, birth injury, convulsive seizures or mental deficiency. This suggests that in approximately 60 per cent of patients with focal EEG abnormality there is some organic pathology which warrants further clinical examination in order to establish the etiologic factors and to institute suitable therapy. In 19 cases, or 40 per cent, who also had an EEG focus the etiology remained obscure. In our series the number of cases with organic factors is in agreement with that of other investigators who reported on diffuse abnormalities in the EEG of children with behavior difficulties (8, 2, 12).

Focal EEG abnormality in behavior disorders as stated above has also been reported: Strauss, Rahm and Barrera (17) described without mentioning the area, "focal dysrhythmia" in seven patients, or 16 per cent, of a series of 44 cases, in 3 of which focal EEG findings were in conformity with organic pathology indicated by neurological examination. Miller and Lennox (12) reported bi-occipital slowing in 6 and unilateral focal (area not mentioned) slowing or spikes in 9 out of 160 patients. Jasper, Solomon, and Bradley (8) noted a "marked bilateral asymmetry" in some cases but did not attempt to analyze their focal findings. Hawkes and Roark (6) found two cases of focal abnormality and some with "inverted sharp waves over the occipital areas" in 25 children with behavior problems. Walker and Kirkpatrick (19) noted "occipital dysrhythmia" in 3 out of 10 of their cases. Schwade and Geiger (15) reported bursts of 6–14 p.s. positive spikes mostly confined to one temporal and one occipital area on the same side of "all patients in a 4-year study of a series of both children and young adults" with severe behavior problems. The similarity of focal waves to those found in EEG's of some epileptics has been mentioned by some of the above investigators.

In the present series, focal discharges in 27 (58 per cent) of the 47 cases occurred in the bi-occipital regions; in 10 cases (21 per cent) in one temporal lobe; in 7 (15 per cent) discharges shifted between the temporal lobes; and of the remaining 3 cases (6 per cent) 2 showed motor

and occipital, and 1 bi-frontal bursts. No significant correlation in the location of focal pattern in the "organic" as compared to the "nonorganic group" was noted. Types of abnormal focal waves were either abortive 3 p.s. spike-waves, high voltage diphasic or negative alpha spikes or bursts, theta bursts with positivity or high voltage serial delta waves. All these types of discharges from various areas are known to occur in epileptics. The abortive spike-wave formations in both occipital regions are indistinguishable from the spike-and-waves which are seen in the same areas of some epileptics who do not have behavior difficulties. Positive or negative alpha and theta bursts from the temporal lobes have been associated with psychomotor or "temporal lobe" epilepsy. However, according to Penfield and Kristiansen (14), approximately 20 per cent of patients with "temporal lobe" epilepsy have an EEG focus in the occipital or frontal rather than in the temporal lobe. In other words, these EEG abnormalities occur in epileptics as well as in children with behavior problems who may or may not have an associated convulsive disorder.

Generalized or focal convulsive attacks in children and adults can in some cases be controlled by anticonvulsant medication. In a study made in this laboratory (13) 6 out of 7 cases of this type were improved by treatment with drugs; EEG records repeated after periods of 3 months to 5 years also showed subsidence of the original delta or spike-wave focus in temporal lobe. Similarity of the EEG pattern in cases with behavior disorder and epileptics does not necessarily imply similarity of the pathophysiological mechanism. Nevertheless, since seizures can be controlled by anticonvulsant medication with improvement in EEG, it is possible that behavior disorders with similar EEG findings can also be benefitted by treatment with drugs, as demonstrated by cases 3, 4, and 5. Such treatment need not necessarily replace usual methods of treatment of these cases.

In a previous study two of us (R. W. W. and B. K. B.) reported generalized paroxysmal fast or slow bursts in 22 adult cases of paroxysmal convulsive disorder "variants" which were characterized by one or more symptoms: unrelieved dizziness, headache, attacks of unreasonable anger or assaultive behavior, psychoneurotic traits, abdominal discomfort with no apparent somatic etiology. Nineteen patients were improved by various anticonvulsant medications combined with psychotherapy (18). Therefore, it appears that some paroxysmal conditions other than convulsions do respond to anticonvulsant treatment. Similar results were also reported by other authors who nevertheless indicate the variability of response to such treatment (19, 4, 1, 11).

Summary

1. Forty-seven cases, or 12 per cent, of 390 children with disorders of behavior showed focal EEG abnormality of various types and in different locations.

2. Bilateral discharges from the occipital lobes occurred in 27 (58 per cent) of the 47 cases; a unilateral temporal focus was present in 10 cases (21 per cent); a shifting temporal focus in 7 (15 per cent); motor and occipital bursts were found in 2 cases; and bifrontal bursts occurred in 1 case.

3. Twenty-eight, or 60 per cent, of the 47 cases with focal EEG abnormalities had some organic etiological factor. In 19, or 40 per cent, no contributory organic factors were present.

4. A trial of anticonvulsant drugs is suggested in selected behavior disorder cases with EEG focus.

BIBLIOGRAPHY

1. Bradley, C.: Problem children: Electroencephalographic diagnosis and pharmacologic treatment. *Connecticut State M. J., 6:* 773–777, 1942.
2. Brill, N. Q., Seidermann, H., Montague, H., and Balser, B. H.: Electroencephalographic studies in delinquent and behavior problem children. *Am. J. Psychiat., 98:* 494–498, 1942.
3. Brown, W. T., and Solomon, C. I.: Delinquency and the electroencephalogram. *Am. J. Psychiat., 98:* 499–503, 1942.
4. Cutts, K. K., and Jasper, H. H.: Effects of benzedrine sulfate and phenobarbital on behavior problem children with abnormal electroencephalograms. *A. M. A. Arch. Neurol. & Psychiat., 41:* 1138–1145, 1939.
5. Gottlieb, J. S., Knott, J. R., and Ashby, M. C.: Electroencephalographic evaluation of primary behavior disorders in children: Correlations with age, sex, family history and antecedent illness or injury. *A. M. A. Arch. Neurol. & Psychiat., 53:* 138–143, 1945.
6. Hawkes, C. D., and Roark, M.: Electroencephalograms of behavior problem children. *EEG Clin. Neurophysiol., 3:* 105, 1951.
7. Henry, C. E.: Electroencephalograms of normal children. Monograph of the Society for Research

in Child Development, Vol. IX, National Research Council, Washington, D. C., 1944.
8. Jasper, H. H., Solomon, P. S., and Bradley, C.: Electroencephalographic analysis of behavior problem children. *Am. J. Psychiat., 95:* 641–658, 1938.
9. Kennard, M. A., and Willner, M. D.: Significance of paroxysmal patterns in electroencephalograms of children without clinical epilepsy. *Res. Publ., A. Res. Nerv. Ment. Dis., 26:* 308–327, 1947.
10. Lindsley, D. B., and Cutts, K. K.: Electroencephalograms of "constitutionally inferior" and behavior problem children; comparison with those of normal children and adults. *A. M. A. Arch. Neurol. & Psychiat., 44:* 1199–1212, 1940.
11. Lindsley, D. B., and Henry, C. E.: The effects of drugs on behavior and the electroencephalograms of children with behavior disorders. *Psychosomat. Med., 4:* 140–149, 1942.
12. Miller, C. A., and Lennox, M. A.: Electroencephalography in behavior problem children. *J. Pediat., 33:* 753–760, 1948.
13. Noshay, W. C.: Subsiding electroencephalographic foci in convulsive disorders. Presented before American Psychiatric Association, Detroit, 1950.
14. Penfield, W., and Kristiansen, K.: *Epileptic Seizure Patterns*, pp. 65, 69 et al. Springfield, Ill.: Chas. C Thomas, 1951.
15. Schwade, E. D., and Geiger, S.: Impulsive-compulsive behavior disorders with abnormal electroencephalographic findings. *EEG Clin. Neurophysiol., Supplement III:* 60, 1953.
16. Secunda, L., and Finley, K. H.: Electroencephalographic studies on children presenting behavior disorders. *New England J. Med., 226:* 850–854, 1942.
17. Strauss, H., Rahm, W. E., and Barrera, S. E.: Studies on a group of children with psychiatric disorders. I. Electroencephalographic studies. *Psychosomat. Med., 2:* 34–42, 1940.
18. Waggoner, R. W., and Bagchi, B. K.: Paroxysmal convulsive disorder variants. *Res. Publ., A. Res. Nerv. Ment. Dis., 26:* 328–331, 1946.
19. Walker, C. F., and Kirkpatrick, B. B.: Dilantin treatment for behavior problem children with abnormal electroencephalograms. *Am. J. Psychiat., 103:* 484–492, 1947.

NEUROPSYCHIATRIC INSTITUTE
UNIVERSITY OF MICHIGAN
ANN ARBOR, MICHIGAN

A DEFENSE ATTITUDE ASSOCIATED WITH IMPROVEMENT IN SCHIZOPHRENIA AND MEASURABLE WITH A MODIFIED RORSCHACH TEST[1]

ZYGMUNT A. PIOTROWSKI, PH.D.[2]

In a lecture on deterioration Nolan Lewis (1) remarked: "Apparently as long as an active conflict is present, deterioration as such does not occur, but when the personality finally finds a satisfactory level in regression, the original mental functions tend to undergo a disuse atrophy. Does a cessation of active conflict indicate the onset of deteriroation? Are they related in any way? Why do we always fear and predict an early deterioration when the patient becomes satisfied at a lower level of adjustment?" In the hope of contributing toward the solution of these questions, this simple investigation was undertaken.

The so-called diagnostic alpha formula (6) for use with the Rorschach perceptanalytic method applies only to that minority of schizophrenics who are overcontrolled in their overt motor behavior and in their social interactions. In these patients there is a diminution if not a cessation of active conflict, and most of these patients seem satisfied with a lower level of achievement and striving. The specific aim of this investigation was to see if the diminution of conflict, associated with a readjustment at a lower level of achievement and efforts, related to deterioration.

There are three different ways in which the Rorschach (8) can be used as a diagnostic aid. They are not mutually exclusive but, on the contrary, complement one another. The first can be called pathognomonic. There are four Rorschach perceptanalytic signs pathognomonic of schizophrenia and two possibly pathognomonic signs of organic cerebral lesions, Plx (perplexity) and Imp (impotence). If strictly defined (8, 3, 7), they are of a very high degree of validity. However, only a small proportion of schizophrenics produces the pathognomonic signs, and for this reason the usefulness of the signs is very limited. The second diagnostic procedure can be called tabular because it is based on components (scoring symbols, factors) which are tabulated and provide a summary of the patient's Rorschach responses. This tabular approach leads to valid conclusions in extreme and clinically obvious cases and fails where it would be of greatest help, viz., in the mild and borderline cases. The tabular diagnostic criteria are set up of average frequencies with which each component occurs in the different diagnostic groups of neuropsychiatric patients. Each component is counted singly and then juxtaposed with other components, the average frequency of which has been established also by noting their frequencies independently of the set or pattern of components in which they occurred. The frequencies of the components so established vary much more with the patient's pre-psychotic personality than with the kind of the disease, psychosis or neurosis. The tabular diagnostic approach has received the greatest degree of attention in literature, but it yielded no significant results, failing especially in the group of diagnostically borderline or most difficult cases. The third approach can be called systematic because it uses patterns of components and because these patterns have been constructed on the basis of a systematic theory of schizophrenic symptoms.

The theory (4) underlying the systematic diagnostic perceptanalytic approach states that if a patient manifests a significant disproportion between his capacity for energy output in interpersonal relationships and his capacity for control over overt social behavior, he is suffering from a mental disorder more serious than a psychoneurosis. When there is a disproportion of this sort, then there are two possibilities: either the energy output exceeds the capacity for control, or the capacity for control exceeds the capacity for energy

[1] This investigation was supported by a grant from the National Association for Mental Health from funds afforded by the Northern Division of the Ancient Accepted Order of the Scottish Rite 33°.

[2] From the Research Division of the New Jersey Department of Institutions and Agencies, Box 1000, Princeton, N. J.

output. The so-called formula alpha applies to the overcontrolled cases. Schizophrenics form only one of several groups of patients whose control capacity exceeds their energy output capacity. Some organic cerebral patients and many manic-depressive depressed psychotics also manifest a disproportionately greater control than energy output. This communication is concerned solely with schizophrenics.

If there is no schizophrenia without some defect in the patient's standards of reality, and if the Rorschach is a valid diagnostic test, then Rorschach records of schizophrenics should contain signs of primary intellectual defects. A standard of reality is defective when (a) the patient's perception is delusional, (b) his reasoning inconsistent and illogical, or (c) both, so that, despite a satisfactory native intellectual endowment and adequate education, the patient's observations and judgment are at marked variance with what healthy people of similar intelligence and education consider real and valid. Now a number of mild although usually chronic schizophrenics produce Rorschach records without any directly noticeable signs of defective reality standards. As a rule these patients are overcontrolled, inactive, and emotionally flat, and some of them display clear primary thought disturbances in clinical examinations. A follow-up of more than five years corroborated the diagnosis of schizophrenia in these cases. Thus arose the question of the validity of the Rorschach perceptanalysis as a diagnostic aid.

The basic principle underlying the systematic diagnostic Rorschach approach explains the absence of direct signs of primary thought disturbances by assuming that the thought disturbances responsible for the defective standards of reality were not given an opportunity to manifest themselves during the Rorschach examination because the patients restricted their mental activity to such a high degree that it enabled them to hide the intellectual defects. The greatly decreased mental, social, and physical activity reduced the chances of manifesting the intellectual defects just as it reduced the manifestations of many latent assets. The principle which seems to be active is that the less is attempted, the fewer errors are made. By analogy, a person with a weak knee may move about without attracting anybody's attention to the defective leg as long as he moves about little and slowly; in such cases locomotion becomes noticeably deficient the moment the patient attempts to walk rapidly or to execute some strenuous movement. As long as the schizophrenic does not tax his mental powers, he may fail to reveal his specific schizophrenic intellectual weaknesses. We know that schizophrenics are terrified by their awareness of their own thought disturbances (hallucinations, delusions, thought blocking, etc.), and that in the early or mild phases of the psychosis the patients are conscious of their primary thought defects, which they try to hide and to rationalize away. Only advanced and markedly deviated patients become unaware of the defectiveness of their thinking because of lack of insight into the nature of their illness. Furthermore in the mild phase of the psychosis, the thought disturbances are not present all the time but make their appearance intermittently and usually when the patient is under considerable stress. The fact that these peculiarities of thought, perception, and judgment appear when the patient is under emotional tension frequently leads to a misdiagnosis of hysteria or neurosis in the mild schizophrenic with whom it is possible to establish a fairly good emotional rapport. It is conceivable that a constriction of mental and other activities may enable the schizophrenic to keep his genuine and relatively mild intellectual peculiarities hidden from the view of others.

The ability to restrain activity and social contacts for the sake of decreasing the chances of manifesting severe personality defects implies an awareness of social implications of one's actions and a desire to appear normal. These traits are incompatible with a severe degree of deterioration. The overcontrolled behavior, as measured by the alpha scale, presumably would not have been initiated by the patient (unconsciously) without some deterioration, but it appears to be, by itself, a compensatory mechanism, designed to hide whatever deterioration there is. Thus the regression to a lower level of functioning and the diminution of activity are related to deterioration in a complex way. If this explanation of the assumed psychological mechanisms responsible

for the development of the overcontrol is correct, then an increase in the alpha score, which is synonymous with an increase of control, should be accompanied by an improvement in the over-all adjustment of the schizophrenic patient. On the other hand, a drop in the degree of control, i.e., a drop in the alpha score, should be accompanied by a worsening in the patient's mental condition. These inferences are based, among others, on the assumption that schizophrenia is a lifelong process but that it can vary in intensity greatly and need not prevent a patient from functioning outside of a hospital, supporting himself, and conducting himself inconspicuously. Should we find that the mental condition of the schizophrenic deteriorates as his control over overt motor behavior decreases, we would be in possession of evidence indicating that the overcontrol is a desirable defense mechanism in schizophrenia. Moreover, should we find that the few neurotics with high alpha scale scores (about 10% to 15% of all high alpha score cases) improve as their alpha scores decrease, and that these neurotics get worse as their alpha scale scores go up, we should have an indirect but pertinent evidence for the belief that there is a qualitative difference between schizophrenia and psychoneurosis. Of course, some schizophrenics may have neurotic symptoms. In spite of the appearance of neurotic and schizophrenic symptoms in the same patient, there may exist a definite qualitative difference between psychoneurosis and schizophrenia.

TABLE 1.—Relation of Change in Alpha Scale Scores to Change in Clinical Status in 50 Adult Schizophrenics after an Average Follow-up Period of 5.6 Years

Changes in Alpha Scores	Clinical Status on Follow-up when Compared with Status at Time of First Rorschach Examination			
	Improved	Essentially same	Worse	Total
Rise in score of more than 1 point	11	1	1	13
Change not greater than 1 point	2	8	6	16
Drop in score of 1 to 3 points	3	—	7	10
Drop in score of more than 3 points	1	—	10	11
Total	17	9	24	50

$\chi^2 = 35.38$ df = 5 $P < .01$

Fifty adult schizophrenic patients, 33 men and 17 women, were reexamined with the Rorschach test after an average interval of 5.6 years. The follow-up period ranged from one to thirteen years; the follow-up period for the middle fifty per cent of patients ranged from two to nine years. Table 1 indicates the distribution of these cases in terms of change in clinical status in the follow-up period, and in terms of changes in the alpha scale score. Most of the changes in clinical condition were evaluated by Dr. Nolan Lewis in connection with another study (6). Clinical status was evaluated on the basis of changes observed in these three areas: (a) Number and acuity of symptoms, including the patient's subjective feeling of psychological comfort; (b) work record and adjustment to family; and (c) level and quality of intellectual processes. If an improvement in (a) was accompanied by a worsening in (c), the patient was considered to have become worse clinically. As is apparent from table 1, in these schizophrenics rise in the alpha score was associated with improvement, while a drop in the alpha score was associated with getting worse. The middle group comprises all the patients whose follow-up alpha score did not differ from the first alpha score by more than one point. In this group of no appreciable change in the alpha score, it was frequently difficult to decide whether or not the patient had undergone any change in clinical status during the follow-up period. Even when the direction of the change was clear, it tended to be small and inconspicuous. This is reflected in the large percentage of patients with the classification "essentially same" on reexamination. These findings are statistically highly significant. This suggests that they can be reproduced by others, provided all essential conditions of the investigation are reproduced. One of these essential conditions is the scoring of the Rorschach records according to the system described in the Compendium (5). The concept that psychosis, a condition more severely pathological than psychoneurosis and characterized by defective standards of reality

testing, is a mental illness in which there is a disproportion between capacity for energy output and capacity for energy control, could not be demonstrated in perceptanalysis without the Compendium modifications of the Rorschach scoring system. Another important condition which must be met to assure repetition of these results is the clinical psychiatric diagnosis of schizophrenia. The 50 schizophrenics of this investigation were diagnosed by Nolan Lewis according to his criteria (2). A significant deviation from these diagnostic criteria will affect the results.

The Rorschach test throws light upon those personality traits which become manifest in the individual's social relationships with others (5). The results of this investigation of schizophrenics show that there is a striking tendency to improve or to get worse clinically according to whether there has been a rise or a drop in the inhibitory control over overt social conduct. Few psychoneurotics receive high alpha scores pointing to overcontrol. Eight neurotics with a high alpha score on at least one of the two Rorschach examinations were studied. In this neurotic group, unfortunately too small for dependable results, the association between changes in the alpha scale scores and clinical changes was the reverse of that observed among the schizophrenics. Among these eight psychoneurotics clinical improvement was invariably associated with a drop of more than one point in the alpha score, and worsening of the condition was invariably associated with a rise in the alpha score of more than one point. This qualitative difference between the schizophrenics and neurotics is easily explained. If neurotics are individuals whose reality testing and reality standards are intact, and who suffer from no primary thought disturbances but only from inhibitions (assumed to be due to a clash between incompatible action tendencies of about equal strength), then a decrease in inhibition or control in a neurotic implies a weakening of the conflict responsible for the neurosis. This lessened conflict would be expected to lead to an amelioration in the patient's clinical condition. On the other hand, an increase in inhibition or overcontrol in a neurotic implies an intensification of the conflict with a subsequent worsening of the clinical condition. Since for schizophrenics a rise in inhibitory control improves the condition and a drop in control makes the condition worse, it can be argued that this control is an essential factor in the patient's handling of his social relations and of his work output. Excessive control (excessive, that is, in terms of the amount required to balance the emotional drives) is evidently not needed by the neurotics as it is by the schizophrenics. The control seems to be an asset to the schizophrenic because it reduces the interference and the potency of a noxious factor. In all probability this noxious factor, as indirectly measured by the alpha scale, is the defective reality standard which differentiates the schizophrenic from the neurotic.

The fact that overcontrol in schizophrenics weakens as the schizophrenic condition grows worse, that is, as the patient's level of psychological functioning continues to drop below the pre-morbid functioning level, supports the assumption that overcontrol cannot be considered a sign of deterioration. On the contrary, the results suggest that the overcontrol implied in high alpha scores indicates good resistance against schizophrenia.

The efficacy of the scale is demonstrated not only by the patients whose scale score rose or dropped, but also by those whose alpha scores remained practically unchanged. In the "no change" group it was frequently difficult to evaluate the change in clinical status because these changes were small or because there was improvement in one area and worsening in another. Nine patients were classified as clinically "essentially same"; the alpha score of eight of them changed by no more than one point and that of the ninth one changed by only two points. Thus there is evidence to support the statement that the alpha scale is a quantitative scale and that its three or four different degrees correspond to different, clinically meaningful and discernible changes in the patient's subjective condition and objective achievement.

In other words, in the high alpha schizophrenics the diminution, if not the cessation, of a conscious conflict is brought about by an over-all reduction in activity, but this does not represent deterioration. These patients regress in many ways and feel more comfortable

functioning on a lower level, not because they have deteriorated and are unable to accurately evaluate their position in life but because they feel more reassured about their ability to keep mistakes of thought and action at a minimum. The decrease in overcontrol, whether it occurs spontaneously or as an effect of therapy, makes the alpha schizophrenics' overt behavior and thinking worse.

The excessive control then can be classified as a compensatory defensive mechanism. However, it is unlikely that this mechanism would develop if the patient did not feel threatened by the possibility of making serious mistakes because of a psychotically defective reality testing. In brief, the alpha scale represents one attempt at constructing an objective and quantitative measuring scale of defense attitudes against the threat of deterioration in schizophrenia.

Table 2 contains the formula which was used to compute the patients' alpha scores. This is a slight modification of the 1950 formula (6). The W column is now somewhat more heavily weighted; the highest possible score for producing no W, or whole responses, was 4 and it is now 5. The Σc–ΣC column was extended at the upper end by the addition of one more step; in the modified formula a patient can obtain 4 points when the sum of light shading responses, the Σc, exceeds the sum of color responses, the ΣC, by more than 4 points. Whenever ΣC is larger than the Σc, the weighted score is zero. Negative weighted scores which are to be subtracted from the positive weighted scores in computing the alpha score are possible only on the ΣC column.

TABLE 2.—DIAGNOSTIC RORSCHACH FORMULA (ALPHA) FOR PATIENTS WHOSE ENERGY CONTROL CAPACITY IS GREATER THAN THEIR POTENTIAL ENERGY OUTPUT

Equivalent Weighted Score	Raw Scores				
	W	ΣC	Σc–ΣC	c′ shock	F + % < 70
5	0	....	....	....	....
4	1	....	>4.0	....	....
3	2	0	3.5–4.0	....	....
2	3	0.5	2.5–3.0	....	....
1	4	1.0–1.5	1.5–2.0	present	present
0	>4	2.0–2.5	0–1.0	....	....
−1	....	3.0–4.0	....	....	....
−2	....	>4.0	....	....	....

Note: For explanation of scoring symbols see reference (5). The alpha scale score is the algebraic sum of all weighted scores received by the patient; see reference (6) for evidence of validity.

It is important to keep in mind that the alpha formula applies only to those patients who produce Rorschach records with (a) a number of whole responses below seven, and (b) a sum of light shading responses at least as large as the number of color responses. Only when both conditions (a) and (b) are met, are the low alpha scores as well as the high alpha scores valid. Any patient with a score of three points or more is said to have a high alpha score; with less than three, a low alpha score. When both conditions (a) and (b) obtain, the chances are 86 to 90 out of 100 that if the alpha score is high, the case is more serious than one of mere neurosis and that, if the alpha score is low, he is not a psychotic or a cerebral organic patient but a neurotic at worst. However, if only one of the two conditions is met, either (a) alone or (b) alone, then only the high alpha scores are valid: in such cases the low alpha scores do not argue against the possibility of a psychosis or intracranial pathology; on the other hand, the high alpha scores retain their validity in indicating a psychosis or brain damage. Reference should be consulted (6) for additional differential diagnostic perceptanalytic criteria which aid in the discrimination between schizophrenics and organic cerebral cases when the alpha score is high. An alpha score of at least five points excludes almost certainly the possibility that the patient is merely a psychoneurotic.

It follows from the structure of the alpha formula that the same and similar alpha scores can be obtained from diverse combinations of components. Each of the components that are constituent parts of the formula is correlated with personality traits which differ greatly from those correlated with the other components. It is therefore possible for the patient to undergo noticeable personality changes without affecting his alpha scale score. However, since the formula measures degrees of control over available potential energy output, it is assumed that the degree of control remains the same if the alpha score remains

stable. The degree of control measured is a relative quantity in the sense that when both energy output and control capacity vary at the same rate, the control measured by the alpha scale will not change.

The average pre-follow-up alpha score of the 13 schizophrenics whose alpha scores rose after the follow-up period was 2.23, while the average pre-follow-up alpha score of the 37 schizophrenics whose alpha scores remained the same or dropped on follow-up was 4.38; there was no significant difference between the averages of those whose scores did not change and of those patients whose scores dropped. The reason for the difference mentioned above is that many of those whose scores rose to reach the value of at least 3 points on follow-up were undercontrolled at the time of the first Rorschach examination; in technical terms, many of these patients were not alpha cases at first. Thus improvement is associated not only with a rise in the alpha score but also with shifting from the non-alpha to the alpha group provided the patient is a schizophrenic. This is an added piece of evidence indicating that the type of overcontrol measured by the alpha formula is a desirable defense attitude designed to minimize the effects of psychosis upon the social behavior of the schizophrenic patient.

BIBLIOGRAPHY

1. Lewis, N. D. C.: (Lectures in N. Y. Psychiatric Inst.), 1940.
2. Lewis, N. D. C.: Criteria for early differential diagnosis of psychoneurosis and schizophrenia: *Am. J. Psychotherapy, 3:* 4, 1949.
3. Piotrowski, Z. A.: The Rorschach inkblot method in organic disturbances of the central nervous system. *J. Nerv. & Ment. Dis., 86:* 525, 1937.
4. Piotrowski, Z. A.: Experimental psychological diagnosis of mild forms of schizophrenia. *Rorschach Res. Exch., 9:* 189, 1945.
5. Piotrowski, Z. A.: A Rorschach compendium, revised and enlarged. *Psychiat. Quart., 24:* 543, 1950.
6. Piotrowski, Z. A., and Lewis, N. D. C.: An experimental Rorschach diagnostic aid for some forms of schizophrenia. *Am. J. Psychiat., 107:* 360, 1950.
7. Piotrowski, Z. A., and Lewis, N. D. C.: A case of of stationary schizophrenia beginning in early childhood with remarks on certain aspects of children's Rorschach records. *Quart. J. Child Behavior, 2:* 115, 1950.
8. Rorschach, H.: *Psychodiagnostics, a Diagnostic Test based on Perception,* pp. 226. Berne, Switzerland: H. Huber, 1942.

NEW JERSEY NEUROPSYCHIATRIC INSTITUTE
BOX 1000, PRINCETON, N. J.

THE MECHANISM OF GOAL ORIENTATION AND THE MANNER OF ITS DISRUPTION

NINA BULL[1]

I. The Mechanism

In the present study, which is an outgrowth of the attitude theory of emotion (1),[2] a mechanism will be offered to account for the directional character of purpose and aim—a mechanism on which the most intelligent functioning of man must necessarily depend; and whose interruption is the point of entry for frustration, with all its variations and consequences.

Since there is no concept in general circulation to describe the particular aspects of purpose with which we are here concerned, the term *goal orientation*, already employed to good effect by Maier (9), will be used as having a clearly directional significance to start with and nothing in the way of controversy attached to it.

An essential characteristic of purposive activity as generally understood is that it strives for some ultimate objective more or less distant, and not too easily attainable. This ultimate objective we shall call the goal of the striving, and it is always in the last analysis designed to satisfy some basic need of the individual, *per se* or as a member of the race. In this capacity (of offering satisfaction) the goal is definitely something wanted or desired, for self or for others—something that is positively valued, that is to say "looks good," whether it be an object or a set of conditions.

We propose to use the term *goal orientation* to cover the whole dynamic process of going for (and if necessary searching for) some such desirable objective. Thus goal orientation, whether in the form of attitude or action, is not only purposive but also appetitive and constitutes a recognizable form of aggressional behavior (the "adient"[3] form) in man as in the animals.

The goal as we intend to use the word is never primarily an object of attack (that is of aggression in the "hostile" form) although attack is sometimes incidental to attaining it. Nor is the goal a target at which to shoot, or hurl a missile from a distance. Rather it represents a kind of destination or place of arrival for the activities of the organism as a whole; and this implies that intervening space must be traversed to reach it. Unless the combination of distance and desirability releases some kind of progressional or travelling reaction (incipient or actual) in the organism, the objective will not be called a goal.

Even with the foregoing limitations it is obvious that goals can be of many different types. Not only may a goal be either an object or a situation, but some goals serve as ways and means to others which are final or consummatory. And some goals serve as substitutes for others that are unattainable.

For the time being, however, we are not concerned with different types of goal, but only with certain principles that apply to goals in general—and to reaching them. And since our interest lies primarily with *human* purposes and projects, we shall confine this study to the essential mechanism of goal-oriented purpose as it first developed in the early vertebrates (3) and has continued fundamentally unchanged throughout the series, up to and including man.

The goals of these early vertebrates were reached by means of locomotion with vision as its guide (3), as will be presently explained, and we here submit the thesis that this intermediary locomotion is still an essential factor in all goal-oriented behavior and constitutes the motility component in the mechanism. In line with attitude theory it would be present in the preparatory stage, goal-oriented motor attitude, as an incipient locomotory reaction or readiness to go ahead in the direction of a goal. And since goal orientation implies an intermediary need for travelling, the active progressional phase can be considered as an extension of the preparatory attitude, in space

[1] Director of Research Project for the Study of Motor Attitudes, New York State Psychiatric Institute, New York City.

[2] Attitude theory postulates *feeling* as a feed-back from preparatory motor attitudes. It is applicable to all behavior where feeling is a factor.

[3] Adient is the term introduced by E. B. Holt (5) to describe the act of *going toward*.

as well as time. It is designed to carry an initial readiness for some consummatory action pattern into contact with the distant stimulus (the goal) required to release it and bring the appetitive striving to a satisfactory end.

The concept of two components—appetitive and consummatory—as regularly found in sequences of instinctive behavior was introduced by Wallace Craig in 1918 (4), picked up and pointed up by Lorenz in 1937, and recently elaborated by Tinbergen (1951) who has presented an illuminating theory (12) based on the successive activity of different levels in the central nervous system.[4] According to this contribution from ethology, which is incorporated in the line of thought presented here, the earlier stage of instinctive behavior is variable and adaptive as well as purposive, while more stereotyped patterns are resorted to as the end is neared; the final consummatory act, which is innate, being more rigidly determined still, and indicating a definitely lower level of nervous integration. The variable and adaptive phase represents a higher level, and the whole conception as outlined by Tinbergen provides a neural substrate for the intelligent phase of purposive activity—goal orientation—which is the subject of our study.

With the foregoing concepts in mind it begins to be apparent why goal orientation, which is geared for flexibility—i.e., for seeking and pursuing, for waiting when necessary, and even to some extent for overcoming obstacles—can be continued fruitfully over long periods of time and can contribute to a satisfying way of life for man. Even when the goal itself is not attained, the action toward it generates, despite its flexibility, a sense or feeling of *making progress* and of *having an aim*—the former being here considered as a feedback from the actual tensions of the progressional or locomotory component.[5]

The sense, or feeling, of *having an aim* is closely related to the sense of progress, but another set of tensions is required to account for it. It is here considered as a feedback from those postural and binocular reflexes which yoke the eyes together, hold them steady and cause them to act as one. The binocular mechanism is known to have acquired the power of orienting the body as a whole (3) and was originally developed *in relation to progression* to "head" the animal toward its goal. This steering apparatus constitutes the visual component of the goal-orienting mechanism, according to the present thesis. In fact, the binocular reflexes with their tensional symmetry may be fairly said to have developed in the service of goal orientation in one of its most primitive forms—pursuit of prey; and they are operative when the body is actually moving toward a goal, and also when it is only *prepared* to do so, and holding ready with its forward-facing, forward-looking attitude and aim.

This combination of locomotion and binocularity, subserving motility and aim, is offered as a mechanism to explain the more obvious aspects of goal orientation. It is not, of course, the entire picture. There are other orienting factors present—much more ancient and much less amenable to observation than the visual steering apparatus—and of these the most important for our present thesis is the olfactory steering apparatus on which the motor mechanism of binocularity was grafted to begin with (3). We have already suggested in an earlier publication (2) that the olfactory-gustatory forward drive is operative in human appetitive behavior even when the sense of smell is not involved; and that it is responsible for those vague but agreeable sensations which constitute man's sense of "liking" (in this case for his goal). It leads to "seeking for," which is the root meaning of the terms *appetitive* and *appetite* (Latin *peto*, I seek).

Thus to the sense of *making progress* and the sense of *having an aim* described above, we can add what might be called the sense of *liking*, or general appetitive sense (not by any means confined to food)—all of these being present as feedback in goal-oriented motor attitude and its extension into actual progression.

The essential mechanism of goal orientation as described above is here considered to subserve a normal phase in those various animal behavior sequences that form so large a part of the daily activity of vertebrates below the

[4] This concept has special reference to those innate behavior patterns that subserve such purposes as maintenance and reproduction. It does *not* apply to the emergency patterns of instinctive riddance, defense, etc., which stem from frustration (see section II) and from fear, and as a rule have no preliminary variable stage.

[5] The fact that even an attitude of preparation may give rise to feelings of forward movement was noted long ago by Mach (8) who observed that it is possible to have the sense of moving forward while standing still and thinking about a distant point in space.

human level:[6] There are various modifications to be accounted for, as when some form of labor or manipulation or communication is required, or getting rid of obstacles, as already mentioned, or stopping to "consider" what would be best to do. But we contend that all such modifications are indicative of instinctive behavior in its preliminary variable stage, before the goal is reached to make (the fixed) consummatory action possible. They can be thought of as variations incidental to the main goal-oriented trend which—barring too much interference—is continuous in aim and forward-going tendency, while making temporary shifts in method and sometimes even in direction.

From the present point of view goal-oriented activity in the human species has the same basic sequence of components—first variable and eventually fixed behavior—but the variable stage has come to be further modified and extended through cultural directives, and complicated by the intrusion of interpersonal difficulties often beyond all recognition of its instinctive character and its essential biological aim. The current tendency among certain groups to think of man's behavior as largely non-instinctive can be partly accounted for by this obscuring of the biological objective.

The attempt to make of culture a substitute for instinct has led to a wide-spread instability in human methods of achieving satisfaction, and a resulting propensity to breakdown, by which we mean disruption in the goal-oriented process. This breakdown, or disruption, will be the subject of the following discussion. And as in this present section we have not considered *kinds* of goals, but only principles that apply to goals in general, and to reaching them, so we shall not, in section II, consider *kinds* or *degrees* of breakdown, but confine ourselves to noting certain principles that govern the breakdown process, and pave the way for future study of its endless ramifications.

II. The Breakdown Process

Essential to the thesis we are presenting here is the concept that disruption, or breakdown in the process of goal orientation, begins with disruption in the forward-facing, forward-looking attitude which we have shown as necessary both for the launching of goal-oriented action and for its maintenance. This functional disruption is here considered as the physiological substratum of frustration, which may or may not register in consciousness.

The psychological manifestations of breakdown in its primary form (that is, prior to complications setting in) will necessarily include some sense of blocking or frustration of the drive. But feelings of frustration are often overshadowed by something more specific in the way of affect, such as disappointment, fear for the project, loss of face, sense of inferiority, etc., depending on which aspects of the total situation are in the forefront of awareness. According to our thesis, these negative affective states, together with a great variety of others, can be considered as variations and/or consequences of frustration. They stem from *interruption*, unresolved, and sufficient in its impact to bring *dis*-ruption in the goal-oriented mechanism, and temporarily, at least, prevent the re-establishment of the instinctual forward-facing orientation.

In view of the above, an operational definition of frustration can now be offered: *the disruptive reaction to interrupted goal-oriented action and the consequent development of negative affective states is specifically what is here subsumed under the term frustration.* But interruptions which can be taken by the organism "in its stride" do not produce frustration; and neither does any interruption sufficient in its impact to produce a wholly unrelated reaction, as for example happens in startle and other excessive forms of fear.

The visual changes that accompany frustration (which is normally a transitional state) would logically include some impairment of the binocular reflexes with their essentially straightforward look and aim, and their hook-up with progression, as already described in section I. Actually there is a mounting body of published data (6, 10) pointing to binocularity as not by any means a permanently established visual state, as was earlier believed, but rather as a functional neuromuscular arrangement suitable for certain types of positive activity. And since these reflexes that yoke the eyes together and cause them to act as one are

[6] That a comparable kind of orienting mechanism exists in the prevertebrates was demonstrated by Loeb (7) in his study of tropisms.

known to have acquired the power of orienting the body as a whole, their breakdown or impairment would logically initiate a change in total pattern. Functional dissociation of the eyes can be conceived, in short, as having deep-seated psychological connotations, inasmuch as it implies a breakdown in the integrity of purposive behavior.

The above conception has been already introduced and linked with some preliminary experimental findings, in the author's monograph (1) where it forms the basic postulate in a chapter on *the orienting function of vision.* It was suggested in this chapter that "impairment in visual efficiency . . . may play an important part in giving rise to those unpleasant feelings of obstructed functioning which constitute the experience of frustration."

In a recent study (1954) of visual changes and affective states (11) we have presented evidence of a correspondence between binocular breakdown and psychological disturbance as noted in patients coming for psychotherapeutic treatment. No distinctions between the various types of binocular breakdown were attempted, and no analysis was made of the disturbed affective states, but from the present point of view the latter can all be thought of as frustrational, inasmuch as they include the interruption of binocularity and steadiness, and therefore of goal orientation as a whole.

The main concept presented in this study was that impairment of visual functioning as reported in the findings partly accounts for the feelings of disorientation and resulting inadequacy which are so characteristic of emotional disturbance. It was posited that binocular coordination and visual steadiness are two of the main factors in the orienting mechanism of the body as a whole; and that any malfunction in these areas of oculomotor performance would logically give rise to feelings of disorientation and inadequacy through absence of a clear-cut sense of direction.[7]

In addition to such functional disturbances as the foregoing, and their psychological counterparts as represented by frustration with its train of consequences, there is still the neurophysiological basis of the breakdown process to consider. Disruption of goal orientation means, according to our thesis, a shift to lower levels of neural integration, as happens normally in all consummatory action. But in the case of breakdown, the "stepwise descent" (Tinbergen's phrase) leads to conditions which are commonly called abnormal, one very simple reason being that under such conditions the final action or consummatory stage does not complete the stage of appetitive striving but, rather, interferes with it. The type of conflict thus produced is part of what may well be thought of as the complex of frustration, initiated as this always is by a concern about whatever obstruction is preventing progress toward the goal. This subject will be dealt with in a forthcoming paper on frustration, which will be a continuation of the present study.

Meantime the door is open for an investigation of the functional changes in the central nervous system that underlie frustration—here considered in its essential character as the breakdown of a primary integration, and quite apart from all its complications with their countless autonomic accompaniments.

For such a study to be fruitful, the primary integration, that *precedes the breakdown*, must be envisaged clearly and established as a norm. These conditions we have tried to meet in presenting our concept of goal orientation, with an outline of its mechanism in which the yoking of the eyes plays an important part. We now suggest that in the *un*-yoking, or *dis*sociation of the eyes, which represents a functional split within the steering apparatus of the body, we are provided with a clue to a directional and hitherto neglected aspect of the breakdown process, and one which has significant implications for neurophysiology as well as psychotherapy.

[7] The accompanying olfactory behavior will be explored in a project already under way.

BIBLIOGRAPHY

1. Bull, N.: *The Attitude Theory of Emotion.* New York: Nerv. and Ment. Dis. Monograph No. 81, 1951.
2. Bull, N.: The olfactory drive in dis-like. *J. Psychol. 17:* 3, 1944.
3. Chavasse, F. B.: *Worth's Squint.* Ed. 7, Section I, The Foundations of Binocular Reactions. Philadelphia: P. Blakiston's Son and Company, 1939.
4. Craig, W.: Appetites and aversions as constituents of instincts. *Biol. Bull., 34:* 91, 1918.
5. Holt, E. B.: *Animal Drive and the Learning Process.* New York: Henry Holt and Company, 1931.
6. Kleitman, N., and Schreider, J. E.: Diurnal varia-

tion in ocular performance. *L'Année Psychologique.* Volume Jubilaire: 201, 1951.
7. Loeb, J.: *Forced Movements, Tropisms, and Animal Conduct.* Philadelphia: J. B. Lippincott Company, 1918.
8. Mach, E.: *The Analysis of the Sensations.* Chicago: The Open Court Publishing Company, 1897.
9. Maier, N. R. F.: *Frustration, The Study of Behavior Without a Goal.* New York: McGraw-Hill Book Company, 1949.
10. Michaud, L., and Granier, R.: Mouvements Oculogyres Automatiques et Asynergiques dans les Psychoses Discordantes. *Ann. Méd. psychol., 105:* 288, 1947.
11. Strongin, E. I., Strongin, J., and Bull, N.: Visual changes and affective states. *J. Nerv. & Ment. Dis., 119:* 344, 1954.
12. Tinbergen, N.: *The Study of Instinct.* London: Oxford University Press, 1951.

NEW YORK STATE PSYCHIATRIC INSTITUTE
722 W. 168TH ST.
NEW YORK 32, N. Y.

THE DOUBLE: ITS PSYCHO-PATHOLOGY AND PSYCHO-PHYSIOLOGY

JOHN TODD AND KENNETH DEWHURST

Just as medical men throughout the centuries have sought to explain the psycho-dynamics of autoscopia so, likewise, have men of letters been intrigued with the dramatic possibilities of the double ever since Aristotle described how a certain Antipheron, when taking a walk, saw a phantasmal reflection of himself advancing towards him. It can well be imagined that a visual hallucination of the self is a strange and frightening experience. The victim of this phenomenon suddenly encounters a specular, though animate, image of himself with which he feels indissolubly linked by intimate emotional bonds. In short, he is confronted by his own double in the fullest sense of the term.

It will be seen from a review of the relevant literature that autoscopia has received but scant attention from Anglo-Saxon writers, and that the bulk of research in this field has been undertaken by Continental alienists and neurologists.

Review of the Literature

The etiology of autoscopy has been discussed in considerable detail by Lhermitte (12) and by Hécaen and De Ajuriaguerra (11). These writers have stressed the importance of anxiety, fatigue, attacks of acute labyrinthine vertigo, and the febrile-toxic states of typhus and influenza in the facilitation of *isolated* episodes of autoscopia in otherwise normal subjects. They have also drawn attention to a relatively high incidence of *recurrent* autoscopia in those affected with epilepsy, dementia paralytica, encephalitis lethargica, chronic alcoholism, drug addiction, and schizophrenia. Menninger-Lerchenthal (13) and Hécaen and De Ajuriaguerra (11) have separately discussed the etiological importance of cerebral lesions in general, and of the tempero-parieto-occipital zones in particular. The latter writers have challenged the claim of Menninger-Lerchenthal that cerebral lesions (infective, traumatic, vascular, or neoplastic) associated with autoscopy appear with significant frequency in the *right* parieto-occipital area, as they insist that they are by no means uncommon on the left. Rank (16) has drawn attention to the *narcissism* displayed by authors preoccupied with the double-motif, and suggested that autoscopy involves a projection of the narcissistic libido. Sollier (18) regarded autoscopy as essentially a disorder of coenesthesia, and allocated only a minor role to the visual factor. On the contrary, Dewhurst (9) has remarked on the super-normal powers of visual imagery displayed by many subjects with autoscopia. Bonnier (2) showed that autoscopy may accompany attacks of acute labyrinthine vertigo. Bollea (3) has produced specular hallucinations by electrical stimulation of the parietal cortex.

The authors of this paper believe that, in the past, there has been no serious attempt to distinguish between factors likely to facilitate the appearance of visual hallucinations *in general*, and factors specifically prone to produce visual hallucinations *of the self*. Clearly, there is nothing *specific* about some of the conditions whose association with specular hallucinations has been stressed. For example, addiction to alcohol merely facilitates the occurrence of any kind of visual hallucination and must be classed as a general factor. Narcissism, if proved to be causally related to autoscopy, must be classed as a specific factor since it has no *general* relationship to visual hallucinations. It is therefore proposed to examine in detail some of the factors which underlie the phenomenon of autoscopy, to attempt to separate *specific* from general factors and, wherever possible, to present either clinical or "literary" examples of each type. Moreover, the authors offer a psycho-physiological explanation of the qualitative differences distinguishing one specular hallucination from another (for example, the autoscopic double may be thin and ethereal, or solid and life-like).

I. The Role of Narcissism

Narcissism may take the form of an undue interest in the appearance of one's body, in

one's physical health, or in the workings of one's mind. It is not confined, as is popularly supposed, to an over-weening vanity in one's external appearance. Hence the Beau Brummell is no more narcissistic than the hypochondriac or the profound introspectionist.

Furthermore, it should be stressed at the outset that narcissism may be associated in the same person with other factors (such as supernormal powers of visualization) which are likely to predispose to autoscopia. Such was the case of Gabriele D'Annunzio, the famous lyrical poet, who presented to the world a poignant and impassioned account of his own autoscopic experience with his exquisite "Notturno." Grievously wounded in the right eye as a result of an aeroplane crash in the First World War, the poet returns to an unpretentious apartment in Vienna where he loses himself in gloomy reverie. After a while, he begins to be haunted by a panorama of life-like visions of himself with which he holds an introspective seance: "It is my own face, as seen in a mirror, animated with life, but ennobled by the marks of an age never attained by any mortal creature. From what depths of misery and sin has it come? How many years of bondage have lined that forehead? How many years of servitude have wrinkled that cheek? How many years of weariness have withered those lips?" Later, he witnesses another vision of himself, but this time as a youth of sixteen. He describes the scene as though he were gazing in a semi-detached way at a portrait of himself: "It is the face I had when I was sixteen years old. Behold, all my despair kindles like a shower of sparks thrown up by a heavy blow from a hammer. The forehead is smooth beneath masses of thick, black hair. The eyebrows bear the mark of such innocence that they give to the large, melancholic eyes an expression of indescribable purity. The parted lips of that handsome mouth convey the anxiety of a heart filled with illusions that are about to be destroyed."

From this extract it is quite evident that D'Annunzio gazed upon an hallucination of himself with the fascination of a Narcissus bewitched by his own reflection. That D'Annunzio's hallucination should assume a specular form is scarcely surprising in view of his yearning for beauty and everlasting youth. Antongini (1) (D'Annunzio's biographer) has this to say of his friend: "Self-knowledge has not prevented the Poet from regretting, all his life, that fate did not present him with the body and face of a Foscolo, or Shelley, or a de Musset. No amount of glory, no triumph over a woman in the field of love has ever sufficed to repay him for this aesthetic flaw." Later, Antongini amplified his remarks concerning D'Annunzio's vanity: "He was the perfect dandy. He would not hear of wearing anything which was not imported, and he would not use a perfume which was not outrageously expensive. His requirements were those of a Beau Brummell."

The poet worshipped unashamedly at the shrine of Hebe and was filled with horror at the ravages of age as revealed by the pitiless evidence of a photograph—"I have just received," he writes, "the photograph which was taken yesterday. It is ruthless, for it shows me as I am and my face just as it is. Nevertheless, whilst riding today, I experienced inexplicably youthful sensations." D'Annunzio's autoscopia assumes a form which depicts certain aspects of his narcissism—an aversion to old age, and a love of youthfulness. His specular visions successively express the fulfilment of a fear (old age) and of a wish (retention of youth).

Another writer prone to narcissism is Guy de Maupassant, who was frequently confronted by an apparition of himself. Of his friend Bourget he asked: "How would you feel if you had to go through what I experience? Every other time when I return home I see my double. I open the door and see myself sitting in the armchair. I know it is an hallucination the moment I see it. But isn't it remarkable? If you hadn't a cool head wouldn't you be afraid?"

In de Maupassant's case his specular hallucinations cannot be dissociated from the dementia paralytica from which he ultimately died. His narcissism was, however, apparent when his intellect was still unimpaired. It is depicted in his morbid horror of dying, on the one hand, and in references to his "lady killing" exploits on the other (*vide* his correspondence with Marie Bashkirtseff). That his narcissism assumed megalomanic proportions under the disinhibiting influence of dementia paralytica is well shown by the following inci-

dent: When walking in the asylum grounds at Passy, de Maupassant broke off a branch from a tree which he handed to his attendant with instructions to plant it, for "next year we shall find it sprouting with little Maupassants."

Another narcissistic poet, Alfred de Musset, was confronted by an hallucination of himself just after he had walked through a cemetery in the company of George Sands. "Suddenly, in the moonlight, de Musset caught sight of a man running towards him. As the stranger drew nearer, the poet saw that his face was haggard, his clothes torn, and his hair dishevelled. The man was drunk; as he lurched past, his face contorted in a leer of hatred. At that instant the poet was overwhelmed with terror for the mysterious stranger was *himself*, twenty years older, with features ravaged by debauchery, mouth agape, and eyes aghast with fear."

De Musset's narcissism took the form of extravagant and dandified apparel, a morbid fear of death, and a proneness to identify himself with his literary characters. His natural tendency to hallucinate was increased by his notorious addiction to alcohol. The poet's specular vision appears to have deep psychological significance in that it symbolized guilt over his addiction to drink with all its sinister implications—premature senility and death! The appearance of the apparition was doubtless facilitated by a morbid train of ideas set in motion by the spectacle of the cemetery.

The stories of the German writer, Jean Paul Richter, are impregnated with the theme of a double or "doppelgänger" no doubt inspired by his own autoscopia. When referring to his autoscopic hallucinations he wrote: "I look at him, he looks at me, and both of us hold our ego in horror." That Richter was an introspectionist obsessed with the *concept of his own ego* is evident from an extract from his autobiography (17): "One morning when still a young child," he writes, "I was standing under the doorway and looking towards the woodstock on the left, when suddenly the internal vision. '*I am an ego*,' passed before me like a lightning flash from Heaven, and has remained with me shining brightly ever since; my *ego* had seen itself then for the first time and for ever."

The famous historian, Hippolyte Taine, was like Richter an introspectionist preoccupied with his own ego. This propensity is clearly shown in a letter to a friend (19): "This afternoon I shall pay some official calls; on these occasions, I always feel as if I were a speaking automaton, or rather as if my ego were stationed outside, whilst a carefully dressed gentleman is making bows and polite speeches on my behalf." On another occasion, Taine (20) describes how his double appeared before him in the pattern of a dream. "At the end of a dream too long for narration," he writes, "my own figure appeared seated in an armchair, near a table, in a white dressing-gown striped with black; it turned towards me, and the shock was so great that I woke up with a start."

Bychowski (4) describes a clinical case of incomplete autoscopia in which he stresses the highly developed narcissism of the patient, which facilitated pathological projection of the altered elements of the body image. In addition, there is an obvious fear fulfilment embodied in the specular vision of her changed (wrinkled) face.

The patient was an intelligent woman of 26 who developed obsessional thoughts of an hypochondriacal nature consisting in imaginary changes in her facial features. She became so preoccupied with these thoughts that she eventually stopped working and retired to bed for a whole year. She complained that she did not want people to see her with a face marred by wrinkles. As a child, she had taken pleasure in standing before the mirror combing her hair and examining her face with close attention. She wanted to appear attractive, and worried about the fact that her cheeks were somewhat puffed out, and that she was not good-looking. When she was 20 years old she began to think that she could detect wrinkles in her face. Soon afterwards, she began to be confronted by the image of her own face deformed by wrinkles. Some inner force pushed her towards the mirror, obliging her to look at the "horrible" picture of her own face. When she was not actually looking into a mirror, she was haunted throughout the day by the mirror image of her changed face.

One of us (J. T.) recalls a somewhat similar case of a young married woman who had been referred to a psychiatric clinic in a state of anxiety with mild depressive coloring. She attrib-

uted her symptoms to worry engendered by recurrent specular hallucinations of four years' duration. Her autoscopy had first appeared when she was in a state of great anxiety, being about to enter a hospital for the surgical completion of a miscarriage (she was exceedingly modest and dreaded medical investigations). She was suddenly confronted by a vivid and realistic vision of herself lying in a coffin. Thereafter, she was much troubled by a phantasmal double of herself which faithfully copied all her movements. Thus, when sitting in a chair knitting, she might suddenly observe (a few yards away) an autoscopic double similarly employed. This chimerical double looked solid and life-like; it was usually dressed in the clothing that she herself was wearing, but occasionally affected a dress that she had recently admired in a shop window. Since the onset of her specular hallucinations, she had become acutely conscious of a compulsion to think about herself! "I may be running up the stairs, when suddenly my thoughts switch back to myself. I cannot stop thinking of myself, and my own voice speaks to me, and says such things as 'Buck up! Don't be miserable'." She insisted that she could "hear" her own voice addressing her although it was inaudible externally. Furthermore, since her illness started, she had developed a tendency to "observe" her own thoughts—to visualize in vivid form the ideas running through her mind. It was noticeable that she dressed and arranged her hair with unusual care. She confessed to being fastidious and pernickety about her personal appearance.

As well as the obvious narcissism depicted by the patient's compulsive preoccupation with herself, she also exhibited the phenomenon of "écho de la pensée" and had augmented powers of visualization. Moreover, the mechanisms of fear and wish fulfilment are clearly discernible in this case.

Summary.

The paramount importance of narcissism as a specific factor in the etiology of the autoscopic double is clearly shown in many of these cases. The significance of narcissism in facilitating a specular hallucination is shown *par excellence* in a case cited by Taylor (21) of a homosexual who: "always dreamed of committing mutual masturbation *with a man very similar in appearance to himself*."

It should be stressed that in some of the illustrative cases quoted there is an interplay of factors, for, in addition to narcissism, fear, anxiety, wish fulfillment, or the super-normal power of visual imagery has played a part in projecting the autoscopic double. Whilst super-normal power of visual imagery may be regarded as a factor likely to facilitate the appearance of visual hallucinations *in general* (and will be discussed hereafter), narcissism should be regarded as a *specific* factor facilitating the appearance of visual hallucinations *of the self*. In view of this, the high incidence of autoscopia amongst creative writers is hardly surprising as they frequently possess both these factors as traits of their personality and occupation.

II. The Role of Super-Normal Powers of Visualization

Francis Galton (10) was the pioneer in investigating the degree with which people can recall past sensations and see images in their mind's eye. He noted that visual memory varied greatly. To some people it had no meaning, whereas others were able to revive an early visual experience with hallucinatory clarity. The latter he called "visualisers": at the other extreme are the congenitally blind who never experience visual hallucinations (23).

Amongst creative writers who are known to have witnessed autoscopic hallucinations there are many examples of their super-normal powers of visual imagery which, in some cases, is combined with extreme narcissism. Amongst the writers already mentioned, D'Annunzio, de Maupassant, de Musset, and Richter are good examples of visualizers. Cazzamalli (5) relates how D'Annunzio when composing "Francesca" suddenly shouted to his servants for candles. When they arrived, accompanied by the great Duse, he was heard to shout repeatedly "Malatestino" (a character in "Francesca") and seen to follow with his eyes the movement of some invisible phantasm. In discussing Maupassant's autoscopia, Coleman (6) referred to the writer's astonishing powers of visual imagery: "He could see anything and afterwards visualise it." Both de Musset and Richter were so plagued by a proclivity to

visualize their thoughts that they were often at a loss to distinguish fact from fancy.

The renowned German poet Goethe was also a visualizer. He could conjure up in his mind's eye the clearest image of a flower, and then follow with ease the stages of a complex metamorphosis, involving a bewildering series of panoramic changes in its appearance. One evening when he was riding home, his mind preoccupied with melancholy thoughts, he suddenly saw a horseman galloping towards him wearing a reddish-brown tunic. As the rider drew nearer, he was astonished to see that it was *himself*. Desperately shaking his head to dismiss the apparition, Goethe was greatly relieved to find the specter gone when he again looked up.

Among the "shadows of all forms that think and live," which so often disturbed the serenity of the poet Percy Bysshe Shelley, was to be found his own shadow in the form of a recurrent, specular vision. Shelley admitted to his friends that he had many visions, and that once, in the garden, he had seen the figure of himself stride up to him and ask, "How long do you mean to be content?" There is no doubt that Shelley visualized with hallucinatory clarity; one night, when listening to Byron reciting some lines from "Christabel" about the lady's breasts, he suddenly sprang to his feet, uttered a piercing scream, and fled from the room. He had seen a vision of a woman with eyes instead of nipples!

There are many such examples in literature, but perhaps the importance of super-normal power of visual imagery is best illustrated in the following case (K. D.) of an otherwise healthy subject:

A doctor set out alone on a walking tour. He started in the late afternoon and decided to walk all night. After progressing at a leisurely pace for about three hours, he experienced the visual hallucination of fruit hanging from the branches of apple and pear trees which he well knew were barren at that time of the year. Just before dawn, he sat down to rest by the roadside. Immediately, he saw his own image moving towards him as though it was slowly unfolding itself from the ground. He had a clear view of its face and the upper part of its chest as though "looking into a mirror." The image was vivid, not transparent, and appeared to be about three to four feet away from him. It persisted for about one minute during which time his reaction was one of "mild surprise." He explains his "mild" emotional reaction as being due to the fact that he has always been "a strong visualizer" and had previously experienced vesperal hallucinations and seen eidetic images. In this case the two general factors of fatigue in a visualizer combined to facilitate the phenomenon of autoscopia.

III. The Role of Archetypic Thinking

In the process of dissolution, the central nervous system exhibits a hierarchical devolution, as phylogenetically older mechanisms are progressively released from the control of higher centers. Similarly, archaic modes of thinking are released in the process of the accompanying dissolution of the personality. As a result, bizarre hallucinatory-delusional themes invade consciousness. Visual hallucinations originating in this way may assume any form, but man's ancient preoccupation with his reflection and shadow particularly favors the appearance of the autoscopic double. The myths and religious beliefs of primitive tribes throw much light on the origin and importance of the double-motif in human psychology. Tymms (22), in discussing the origins of the double-theme which figures so conspicuously in the world's romantic literature, refers to the Indian tale of Nala and Damayanti: Four Gods woo Princess Damayanti, but she prefers a mortal, King Nala, for her husband. In order to avenge the slight, the gods assume Nala's form, and sit beside him in a row when Damayanti comes to pick him out from the throng of suitors. At first she is baffled and confused by the four doubles of her lover, but the gods relent and vouchsafe to her the necessary knowledge to distinguish her human suitor.

Tymms also cites the myth of Amphitryon, whom Jupiter impersonates (as a double) to deceive Alcmena, wife to Amphitryon. The same writer relates how, in the monkish anthology of the Gesta Romanorum, the Emperor Jovinian was punished for excessive pride by being replaced by a "false Jovinian," his double.

Crawley (7) has made an exhaustive study of the role of the double in primitive folk-lore and tribal religions: The individual's soul or

ghost is often believed to have the capacity to act as the double of its host. The soul-double assumes a material form and closely resembles the prototype. Among the Nāgas the "ghost" is an exact image of the deceased as he was at the moment of death with scars, tattoo marks, and mutilations. Furthermore, the ghost requires and desires food and other sustenance.

Andamanese souls partake of the form of the person to whom they belong. The East Indian Islanders believe the soul to be an exact replica of the person; it is a copy or abstract of its living prototype, but is always *material*. Among the Karo Bataks the soul is the copy of the owner—his other self. The Egyptian *ka* is a classic example of these beliefs. The *ka* could live without the body, but the body could not live without the *ka*; yet the *ka* was material. It is represented not only as a miniature duplicate of the person, but sometimes as half the size, sometimes as full size.

Precope (15) sums up the situation thus: "The savage conceives the soul to be either an exact miniature of himself, a microscopic replica lodged in the head, or his own shadow, and regards sleep as the temporary, death as the permanent absence of this miniature or of his own shadow."

There is therefore weighty evidence that the autoscopic double is in the nature of a psychological atavism of profound philosophical interest. Reversion to archetypic thinking is a racial characteristic, but certain individuals are peculiarly prone to indulge in archaic thinking. Archetypic thinking may therefore be regarded as either a general or specific factor, as the case may be.

IV. The Role of Disturbance of Function in Somatognostic Areas

There are sound reasons for believing that epileptic discharges in those areas of the cerebral cortex concerned with the integration of the body image will produce disorders of the latter (hyposchematia, hyperschematia, autoscopy, and the like). An association between autoscopy and lesions in the temporo-parieto-occipital zones has been noted by a number of writers (11, 13, 14). Bollea (3) has electrically stimulated posterior zones of the parietal lobes and produced sensations of elongation of the body image, disappearance of the four limbs, and *autoscopic visions*. Irritative lesions in the posterior parietal regions must therefore be accounted as among the *specific* causes of autoscopic hallucinations. The following case report (K. D.) illustrates the important role of trauma, leading to disturbed function of somatognostic areas, and hence precipitating frequent episodes of autoscopia: A male patient, aged 57, was admitted to hospital complaining of episodes during which he "saw himself." In 1917, his right eye was destroyed by a fragment of shrapnel which passed through the orbit into the right posterior parietal lobe where it is still lodged. The patient has a clear recollection of sustaining the injury, but lost consciousness a few hours later. He had post-traumatic amnesia for four or five days and was in a hospital for a period of six months. In 1922, he first began to suffer from attacks of grand mal about which he has only a hazy recollection. Usually, there was no aura; but on one occasion he did remember gazing at the leaves of a tree rustling in the wind. Gradually they began to change into "little colored goblins" and immediately afterwards he lost consciousness. The patient has been told that during these fits (which usually occur at night) he always has convulsions, froths at the mouth, and often bites his tongue. He has had 10–20 such episodes in 20 years—the last occurring in 1942. The nature of these attacks altered in 1930 when he first began to see a black spot in front of his eye. "I've seen the X-ray of the bit of metal in my head and it looked just like that." The scotoma lasted intermittently for some hours: afterwards he saw the figure of a man. "I was in the doctor's surgery staring into the garden. Then I saw the man about four feet away to my left. It suddenly dawned on me who it was. It was me." These hallucinations of a normal-sized mirror image of himself lasted for about 10 days. His "double" seemed solid, but there was no feeling of affinity between them.

Seven years ago the patient had another curious experience. He was alone in a wood gathering sticks. "I knew that I was alone, yet suddenly I felt surrounded by hordes of people." This feeling, which lasted intermittently for about one week, was not accompanied by any hallucination. In January, 1954 (one

month before admission), the right-sided headaches, which are a sequel of his original injury, became more severe and more frequent. A fortnight later he again experienced visual hallucinations, which for the first three or four days took the form of multicolored lights. Later they became more organized, and he saw "crowds of tiny figures all the colors of the rainbow—*all myself*." These figures approached and then receded. He also saw stairways and other interior parts of a house. "Sometimes it was my own house, other times I couldn't recognize it." Each episode lasted two or three minutes and was preceded by a feeling of nausea. At the time of his admission these hallucinations had become almost continuous; the remissions lasted only a few minutes and even then his vision was distorted. "The house across the road looked much bigger then I knew it to be; and the wireless in the corner of the room looked the most ridiculous thing you ever saw." He stressed the fact that during these latter episodes he was conscious, fully aware of his surroundings, and quite capable of carrying on a conversation. The patient, who farms a smallholding, is a non-drinker and non-smoker. His only past illnesses have been herpes zoster in 1926 and pleurisy in 1942. There is no family history of psychological illness.

Clinical examination revealed a scotoma in the upper quadrant of the left visual field: other systems appeared natural.

Body-image disturbances associated with lesions of the labyrinthe are probably mediated through somatognostic areas. A case is described by Bonnier (2) in which a patient, during an attack of right-sided labyrinthine vertigo, underwent an illusory division into two persons (two bodies). It was as though a second body freed itself from the first, and took up a position to the right of it (but facing a little outwards). This patient likened the phenomenon to the splitting of an image by turning the prisms of a stereoscope.

V. Psycho-Physiological Problems

The individual's "body image" is evolved by a continuous integration of countless, successive impulses—tactile, kinesthetic, visual, and auditory (the sound of the voice). This integration of sensory impulses from the various parts of the body into a body image is accompanied by the development of a complementary psychical component which imbues it with *personal* significance. As a result, the individual is not merely aware of the external appearance, position, and "feeling" of his body, but also bears towards it a sentiment of ownership with all the emotional and ideational accompaniments. This composite body image, formed from the aforesaid neural and psychical components, is liable to undergo partial or complete schism with separation (by projection) of the sequestered portion. On the one hand, the composite body image may suffer complete division with projection of *all* its components; on the other, this process of schism with projection may involve only *one* component. Menninger-Lerchenthal (see Bychowski, 4) believed that specular hallucinations were produced by a projection of both kinesthetic and visual elements from the composite body image. Actually, the autoscopic double varies in its degree of perfection. It may consist of an hallucination of the *whole* of the subject's body, or merely a *part* of it (for example, the face or arm); it may be a solid, lifelike replica, or semi-transparent and vague; it may exchange ideas with its prototype in complete silence, or speak with the voice of the latter; and recognition by the subject of the hallucination as his own double may be gradual or immediate.

Presumably, these differences are brought about by variations in the number and strength of body-image components which have been projected. All the components of the composite body image would have representation in the perfect autoscopic double.

In a case (that of Dr. Hal Gregg, San Francisco) described by Critchley (8), *only* the kinesthetic and psychical components of the composite body image appear to have suffered schism and projection; the absence of the visual component is an intriguing feature of the case. A woman of 43 years, exhibiting a number of neurological signs and symptoms following two seizures, would "wake in the night with the very intense feeling that someone was in the room—a person she knew; indeed, with whom she was very familiar. Sometimes, she was at a loss to decide who this could be, but

on other occasions, it would dawn on her that this person was none other than herself. . . ."

Here, the kinesthetic component seems to have been much stronger than the psychical, hence the tardy identification of the "presence." It is significant that an intrusive feeling of being in the presence of an invisible companion is a frequent experience of those subject to autoscopia.

In addition to the "shrapnel case" already described, in which the lack of a feeling of affinity between the patient and his double suggests an absence of the psychical component of the body image, the writers have personal knowledge of two cases which are pertinent to the issues under examination: In the first case, a young girl of 17 years, a sufferer from migraine and epilepsy, is troubled by bizarre disorders of the body image. In particular, she is prone to lightning attacks of depersonalization of such intensity that she stops in the middle of a sentence to ask abruptly "am I really here?", at the same time pinching her own face in an attempt to supply the answer. This patient occasionally becomes aware of a feeling of having "an invisible double, like a shadow" stationed a yard away on her left. This intangible double seems to her to contain her mind.

This case appears to illustrate the projection of the psychical component of the body image (with depersonalization), and of the psychical and kinesthetic components (with a psychical and kinesthetic hallucination—an invisible double).

In the second case, a woman aged 32, a sufferer from migraine and epilepsy, was troubled by symptoms of a most unusual character. These included distortions of the body image with illusory shortening of the legs, attacks of angor animi, and acute feelings of depersonalization. The latter were so severe that she was at times constrained to look in a mirror to assure herself that she was really present in the room. However, her most singular symptom was the hallucination of a supernumerary phantom arm which developed from time to time. She would first become aware of a feeling that "something" was lying along the anterior surface of the left arm. When she looked to see what it was, she would observe an extra arm and hand lying on top of the real one. This phantom was thinner and slightly shorter than its companion; it was clad in a sleeve, the material being that of her real sleeve. Furthermore, the fingers of the phantom arm moved in unison with flexion-extension movements in her real arm. The phenomenon lasted about half an hour and was so realistic that she would hide the arm behind her back for fear that others might notice the phantom arm, although she really knew that it was but a product of her own fancy. If she attempted to touch the phantom arm, she found that there was nothing there.

Clearly, the supernumerary phantom arm is in the nature of *an autoscopic double of the real arm*, produced, no doubt, as a result of a transient vascular disturbance affecting a portion of the right parietal lobe. A migrainous rather than an epileptic mechanism is favored by the fact that the hallucination lasted some thirty minutes. The fact that she *felt* the phantom arm, before she saw it, points to the presence of *kinesthetic* as well as visual and psychical components. This case, therefore, illustrates *par excellence* the tendency for kinesthetic elements to be projected in the autoscopic process. Here again, the attacks of depersonalization suggest a projection of the psychical component of the body image—the latter endowing the body, or its parts, with the sentiment of personal ownership.

Conclusion

Specific factors facilitating the appearance of visual hallucinations *of the self* can, in the main, be separated from general factors which facilitate the occurrence of non-specific visual hallucinations. Factors, such as narcissism and irritative lesions in the somatognostic areas, play a specific role as etiological agents underlying the autoscopic hallucination. Such a motley group of conditions as anxiety, fatigue, addiction to drugs or alcohol, cerebral lesions (not confined to somatognostic zones), psychoses, etc., are non-specific factors as they merely render the subject prone to visual hallucinations *of any kind*. The enhanced powers of visual imagery displayed by certain individuals must also be accounted a general factor, as these subjects' tendency to visual hallucination is by no means confined to autoscopic hallucinations. Frequently, autoscopia appears to arise from a complementary interplay

of specific and general factors (narcissism and super-normal powers of visual imagery, for example). The role of archetypic thinking may be general or specific in that the tendency to revert to primitive forms of thinking, while fundamentally universal, varies in degree from one individual to another. The specular hallucination may serve as a mechanism for the expression of latent fears or wishes. Experimental and pathological studies point to the parietal lobe as a site of particular importance in somatognostic integration. Irritative lesions (traumatic, vascular, neoplastic, or infective) in the tempero-parieto-occipital zones may be associated with specular hallucinations. Disturbance of function of somatognostic integration areas may be produced indirectly by irritative lesions in the labyrinthe (proprioceptive system); accordingly, autoscopy may occur in association with attacks of acute labyrinthine vertigo. The specular hallucination may be anatomically complete or incomplete; the subject may be confronted by an hallucination of his face or by an hallucination of his whole body. The autoscopic hallucination may be psycho-physiologically complete or incomplete; the complete specular hallucination involving a schism and projection of visual, kinesthetic, auditory, and psychical components of the composite body image. Incomplete forms lack one or more of these four components.

BIBLIOGRAPHY

1. Antongini, T.: *D'Annunzio.* London, William Heinemann, 1938.
2. Bonnier, P.: L'aschématie. *Rev. neurol., 54:* 605, 1905.

Bollea, G.: Contributo Sperimentale alle fisiopatologia del casidetto schema corporeo. *Riv. neurol., 18:* 337, 1948.

4. Bychowski, G.: Disorders of the body image. *J. Nerv. & Ment. Dis., 97:* 310, 1943.
5. Cazzamalli, F.: Creative hallucinations of writers and artists. *Giorn. psichiat. e neuropatol., 65:* 336, 1937.
6. Coleman, S. M.: The phantom double. Its psychological significance. *Brit. J. M. Psychol., 14* (Part 3): 254, 1934.
7. Crawley, A. E.: *Doubles. Hastings' Encyclopaedia of Religion.* London, 1908–1926.
8. Critchley, M.: *The Parietal Lobes.* London, Edward Arnold, 1953.
9. Dewhurst, K.: Autoscopic hallucinations. *Irish J. M. Sc., 1:* 263, 1954.
10. Galton, F.: *Inquiries into Human Faculty and its Development.* London, Everyman, 1883.
11. Hécaen, H., and De Ajuriaguerra, J.: *Méconnaisances et Hallucinations Corporelles.* Paris, Masson, 1952.
12. Lhermitte, J.: *Les Hallucinations.* Paris, G. Doin, 1951.
13. Menninger-Lerchenthal, E.: Quoted by Hécaen and De Ajuriaguerra in (11).
14. Nouët, H.: Hallucination spéculaire et traumatisme cranien. *Encéphale, 1:* 327, 1923.
15. Precope, J.: *Medicine, Magic, and Mythology.* London, William Heinemann, 1954.
16. Rank, O.: *Don Juan. Une Étude sur le Diable.* Paris, Denoel et Steele, 1938.
17. Richter, J. P. F.: *Levana or The Doctrine of Education (with autobiography).* George Bell, 1884.
18. Sollier, P.: *Les Phénomènes d'Autoscopie.* Alcan, 1903.
19. Taine, H. A.: *Life and Letters of H. Taine.* London, Archibald Constable, 1904.
20. Taine, H.: *On Intelligence.* London, L. Reeve, 1871.
21. Taylor, F. H.: Homosexual offences and their relation to psychotherapy. *Brit. M. J., 2:* 525, 1947.
22. Tymms, R.: *Doubles in Literary Psychology.* Cambridge, Bowes and Bowes, 1949.
23. Wagener, H. P.: Visual hallucinations. *Am. J. M. Sc., 215:* 226, 1948.

MENSTON HOSPITAL
MENSTON-IN-WHARFEDALE
YORKSHIRE, ENGLAND

SUDDEN, VERSUS PROLONGED, IMMINENT DEATH
An EEG Comparison

LESLIE J. SEELEY, M.D.

Introduction

Recently the writer (1) reported the EEG findings in a sudden, non-toxic death due to coronary thrombosis with occlusion. Schwab (2, 3) described repeated EEG recordings in a case of a man with a complete section of the cervical cord. Seven hours and 4 minutes after the section, this patient died, compared to 9⅙ minutes after the coronary occlusion, in this case reported.

Impending death recordings from various toxic causes have been reported but will not be considered in this paper.

Technique

Both monopolar (scalp to ear or ears) and bipolar (scalp to scalp) methods of electrode placement were used with a Grass, Model III A, six-channel amplifier. Chloral hydrate, 0.65 gm., was given to induce sleep. The case was for routine examination purposes customary at this hospital.

Results

Within five minutes after the coronary occlusion, the amplitude had decreased from an average of 45 to 11 microvolts (UV).

The rhythm had changed from almost continuous to short bursts of one to two seconds with absence of electrical activity for from one to three seconds (figs. 1 and 2). The frequency had likewise decreased from an average of 9½ cps. to an abnormally slow rate of 6 cps.

Within 6 minutes after the occlusion, the frequency had slowed to 5½ waves per second. The amplitude had dropped to 10 UV, and then only in short bursts, with long periods of non-activity. The brain wave activity (amplitude, frequency of waves, and continuity of rhythm) steadily decreased to the vanishing point (fig. 3).

Discussion

It is obvious that there is a markedly differing pattern of brain-wave change in these two lethal, non-toxic conditions. In the case of sudden death following the coronary occlusion, there was rapid and progressive diminution in amplitude, frequency of waves, and continuity of rhythm. Conversely, in the more prolonged death there was very slight change in any of these three considerations for at least 6 hours after the fatal injury.

Schwab (3) described in excellent detail the EEG recordings of the prolonged, impending death due to cord section, starting 4⅙ hours after the fatal injury. He described the first recording, "EKG and EEG were fairly normal. EEG amplitude 20 UV. Frequency 7–8 cps." There was no previous EEG recording with which to compare.

Six hours and 17 minutes after the injury the EKG remained the same, but all EEG activity was absent. Nineteen minutes later (six hours and 36 minutes after injury, and about ½ hour before death) there were paroxysmal bursts of abnormally slow waves (5 cps.), with no activity between bursts for 1 to 2 seconds. Eleven minutes later (17 minutes before death) there were paroxysmal bursts of bilateral delta (2½ cps.) waves. The EKG remained unchanged.

Three minutes later there was little EEG activity. Nine minutes later (5 seconds before death) there was very little activity in the right, but there was a final burst of alpha activity in the left hemisphere. Seven hours and 44 minutes after the injury, the heart stopped after feebly beating with a very low EKG. The EEG activity and respirations had already ceased. Then there was a complete lapse of brain-wave activity for about 20 minutes although the heart rate and respirations continued unchanged.

Following this lapse, electrical activity again resumed, but paroxysmal in type and very much slower in frequency, with absence of waves from 1 to 2 seconds between bursts. Subsequent to this there was a gradual decrease in amplitude, frequency, and continuity of waves except for a single terminal burst in the left hemisphere 5 seconds before death. Death was doubtless due to shock.

FIG. 1. Normal EEG shortly before coronary occlusion. Alpha frequency 9½ cycles per second (cps.), sinusoidal alpha waves of 45 microvolts average amplitude. Alpha rhythm almost constant.

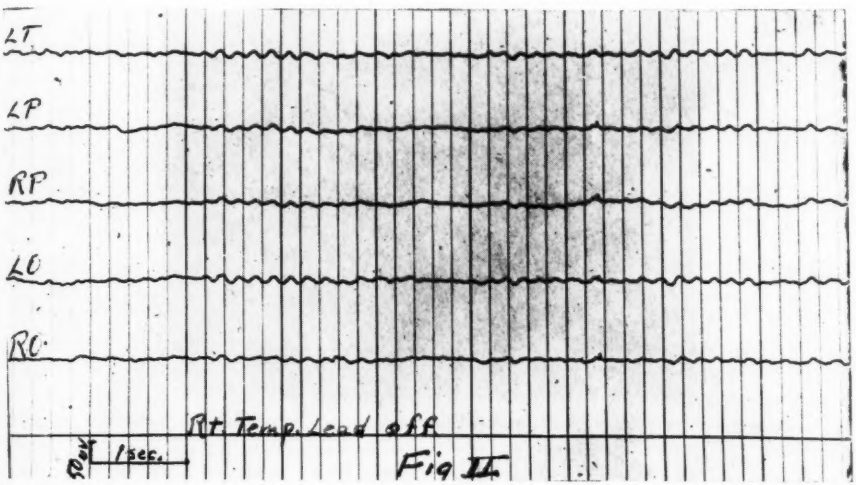


FIG. 2. About 6 minutes after coronary occlusion. Amplitude decreased to 10 UV. Alpha frequency decreased to bursts of slow waves (5½ cps.), with absence of waves between bursts of from one to four seconds.

Despite the coronary occlusion in the first case, it is evident that cerebral circulation, although greatly impaired, was not completely arrested, inasmuch as the electrical activity of the cortex continued for over 9 minutes.

Penfield and Jasper (4) as well as others demonstrated that sudden complete anoxia or ischemia arrests the ability of cerebral neurones to respond before the transient increase in excitability can be manifest in epileptic discharge. Sudden complete arrest of cerebral circulation results in the disappearance of electrical activity of the cortex within *10–15 seconds*.

In the coronary occlusion there was no infarct nor myocardial rupture. The significant findings at autopsy were:

1. Right ventricular myocardium (in diastole) 2 mm. thick: soft and flabby.

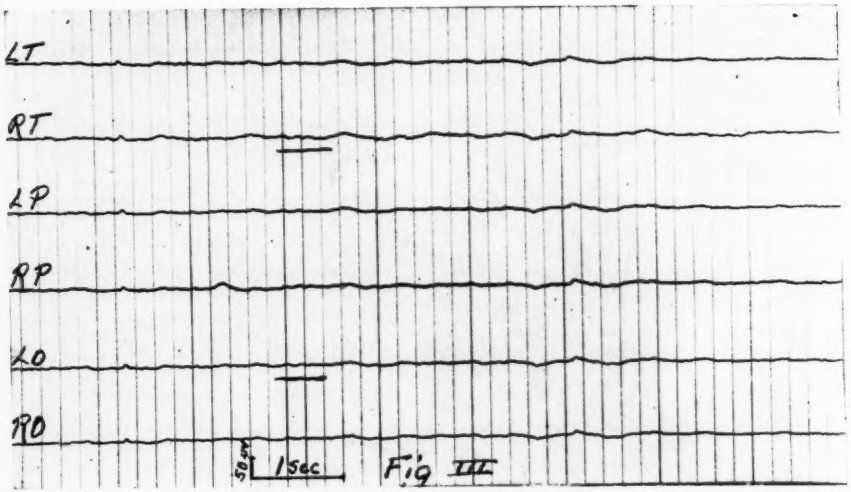


FIG. 3. Nearly complete absence of brain waves, 2⅔ minutes after fig. 2, about 8⅔ minutes after coronary occlusion. This is exactly 30 seconds before death.

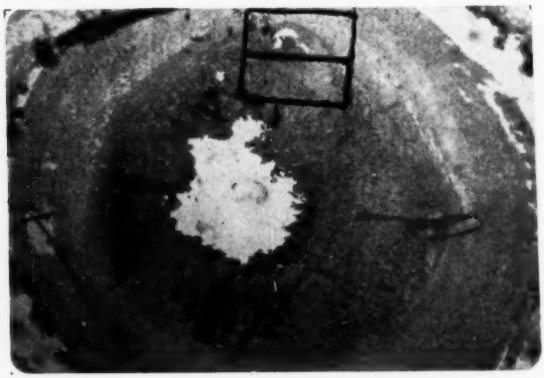


FIG. 4. Coronary artery with pre-mortem clot, hemorrhage into wall, perivascular infiltration and atheromatous wall.

2. Two and one-half (2½) cm. from the ostium of the right coronary artery is completely filled with blood clot. Distal to this there is a narrowing of the lumen and near-obliteration.
3. Microscopic (see figs. 4, 5, and 6) right coronary artery wall shows some atheroma, with lymphocytic and polymorphonuclear infiltration in the periphery and around the vessel. Lumen filled with thrombus.
4. Conclusion: Coronary atherosclerosis, terminating with coronary thrombosis and coronary occlusion.

SUMMARY AND CONCLUSIONS

The contrast of brain-wave changes between a sudden death due to coronary occlusion and a prolonged, inevitable death due to complete section of the cervical cord has been described.

In the case of the coronary occlusion there was a rapid, steady diminution in amplitude

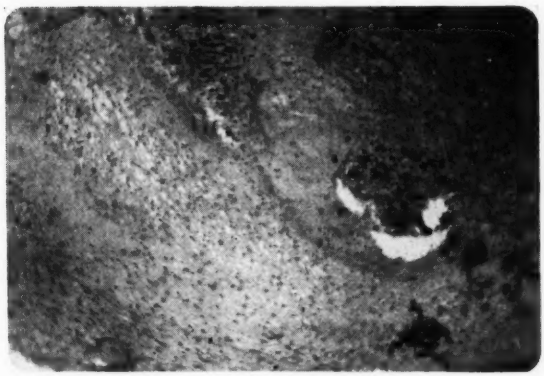


FIG. 5. Clot and portion of wall of Coronary artery. Mid-high power section blocked off in fig. 4.

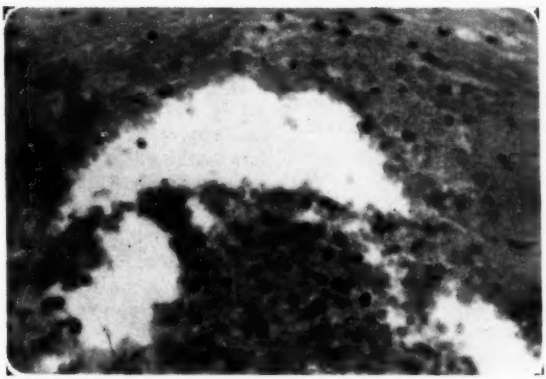


FIG. 6. Portion of upper ½ of blocked-off section in fig. 4. Hemorrhage into wall of coronary artery.

and frequency of waves, as well as the continuity of rhythm, to the vanishing point, 9⅙ minutes after the occlusion.

In the cord-section case the rate, basic, sustained rhythm and amplitude of the brain waves remained relatively normal for over 6 hours, as did the cardiac and respiratory rates. The first of a series of EEG recordings was done 4⅙ hours after the fatal accident (injured 11:40 a.m.; died 6:44 p.m.).

After 6 hours without much change there was a complete cessation of electrical activity from the brain for nearly 20 minutes, despite continued, unchanged heart and respiratory rates. Following this period of absence of brain-waves there were numerous bursts of abnormally slow waves (5 cps.), with periods of 1 to 2 seconds of complete absence of waves. The frequency of waves in these bursts gradually decreased to 2 cps., 17 minutes before death.

All electrical activity gradually decreased in frequency, amplitude, and repetition of bursts until 5 seconds before cardiac arrest when there was a single, terminal burst in the left hemisphere.

These patterns both differ from the early increase of amplitude, frequency, and sustained rhythm in fatal barbiturate coma as described by Talbot (5, 6). (Dr. C. R. McColl supplied microphotographs and autopsy report.)

BIBLIOGRAPHY

1. Seeley, L. J. EEG recording of a death due to non-toxic causes. *J. A. M. A., 156:* 1580, 1954.
2. Schwab, R. S.: The measurement of body currents. *Trans. Am. Inst. Elec. Eng., 60:* 1–5, 1941.
3. Schwab, R. S.: Human EEG and EKG during death. *Electroencephalography in Clinical Practice.* Philadelphia: W. B. Saunders Co., 1951.
4. Penfield, W., and Jasper, H.: *Epilepsy and the Functional Anatomy of the Human Brain,* pp. 227. Boston: Little, Brown & Co., 1954.
5. Talbot, D. R.: Use of EEG to prevent fatalities in barbiturate induced psychoses. *Proc. West. Soc. E.E.G.; EEG & Clin. Neurophysiol., 6:* 347, 1954.
6. Talbot, D. R.: EEG evidence of barbiturate damage to the central nervous system. *Proc. West. Soc. E.E.G., EEG and Clin. Neurophysiol., 1:* 112, 1952.

V.A. Hospital

American Lake, Washington

THE EFFECT OF BENZEDRINE SULPHATE UPON CEREBRAL BLOOD FLOW AND METABOLISM IN MAN

JOHN G. ANDREW, M.D., AND WILLIS SENSENBACH, M.D.

Introduction

The administration of Benzedrine sulfate has been reported to have exerted a stimulating effect upon the psychological processes of emotionally depressed and retarded patients and to have promoted an improvement in their mental efficiency (10); to have increased the intelligence scores of children without "serious mental deviations" (8, 11); to have had no effect upon the mental reactions of mentally retarded children of the moron and borderline classification (3); to have accelerated the intellectual performance of school children (2); and to have been of benefit in the treatment of epilepsy (6).

Little is known, however, of the effect of this drug upon the circulation and metabolism of the brain. As a result of *in vitro* studies on brain slices and minced brain tissue, Mann and Quastel (7) concluded that "Benzedrine owes its stimulating influence to its ability to compete reversibly with amines for the amine oxidase of brain and of other organs, thereby reducing the rate of formation of inhibitory aldehyde," and thus preventing a decline in tissue respiration that otherwise would have occurred. On the other hand, the *in vivo* studies in monkeys of Dumke and Schmidt (4), and Schmidt, Kety, and Pennes (13) have indicated that the intra-carotid administration of Benzedrine reduces both cerebral blood flow and metabolism. Abreu, Liddle, Burks, Sutherland, Elliott, Simon, and Margolis (1), employing the nitrous oxide technique for the determination of cerebral blood flow in man, were unable to demonstrate any consistent change in blood flow or oxygen or glucose uptake of the brain after the intravenous administration of Benzedrine sulfate.

The purpose of the present study was to determine the effect of the oral and more chronic administration of Benzedrine sulfate upon circulatory and metabolic functions of the brain in man.

Clinical Material and Methods

Cerebral circulatory and metabolic studies were made before and after the oral administration of Benzedrine sulfate in 13 young male subjects. One of them (W. F. B.) had diabetes mellitus, which was under control when the studies were made; one (C. L. N.) had early but definite signs of amyotrophic lateral sclerosis; the remaining 11 subjects were either suffering from anxiety or convalescing from an acute non-nervous system illness (table 2) when the studies were done. After completion of the control studies, which were done between 8:00 and 9:00 in the morning with the subject fasting and in the recumbent position, Benzedrine sulfate was administered orally according to the following daily schedule: 10 mgm. at 7:00 a.m., 5 mgm. at 1:00 p.m., and 5 mgm. at 5:00 p.m. After 5 to 11 days, the studies were repeated under the same conditions except that they were done one to two hours after the last 10-mg. dose of Benzedrine sulfate.

Cerebral blood flow (CBF) was determined by the nitrous oxide method of Kety and Schmidt (5), as modified by Scheinberg and Stead (12). Cerebral oxygen consumption (CMR_{O_2}) was calculated from the CBF and arterio-cerebral venous oxygen difference. The cerebral glucose consumption (CMRglu) was calculated from the CBF and the arterio-cerebral venous glucose difference, and cerebral vascular resistance (CVR) from the CBF and the mean arterial blood pressure (MABP). MABP was measured directly from a peripheral artery with a damped mercury manometer. Blood oxygen content and carbon dioxide content were determined manometrically. Blood sugar was determined by Nelson's modification of the Somogyi method (9). Blood samples for pH determination were drawn anaerobically and the pH immediately determined at 37°C. by means of Cambridge Research Model pH Meter. CO_2 tension was calculated from the nomogram of Singer and Hastings (14).

Results

The results are summarized in tables 1 and 2. After the oral administration of Benzedrine sulfate, mean MABP, CBF, CVR, CMR_{O_2},

TABLE 1.—CEREBRAL CIRCULATORY AND METABOLIC FUNCTIONS BEFORE AND AFTER THE ORAL ADMINISTRATION OF BENZEDRINE SULFATE

Subject	Age	Diagnosis	Mean Arterial Blood Pressure (MABP), mm. Hg		Cerebral Blood Flow CBF), cc./ min./100 gm.		Cerebral Vascular Resistance (CVR), mm. Hg/cc./ min./ 100 gm.		Cerebral Oxygen Consumption ($CMRO_2$), cc./min./100 gm.		Cerebral Glucose Consumption (CMRglu mg./min./100 gm.	
			C*	E†	C	E	C	E	C	E	C	E
W. C. P.	29	Anxiety	69	79	88	59	0.78	1.34	5.74	3.66	5.28	7.08
W. L. M.	33	Functional gastrointestinal disorder	81	77	54	58	1.50	1.33	3.41	3.45		
O. W. W.	33	Bronchopneumonia	83	85	64	61	1.30	1.33	3.94	3.77		
J. M. L.	56	Anxiety	82	82	53	65	1.55	1.26	3.06	3.70	4.77	4.55
C. L. N.	29	Amyotrophic lateral sclerosis	80	77	58	68	1.38	1.13	4.35	4.47	5.22	5.44
K. E. H.	34	Anxiety	95	93	82	81	1.16	1.15	3.76	3.47	6.56	0.81
J. L. P.	40	Alcoholic gastritis	92	88	57	54	1.61	1.63	3.76	3.74	7.41	6.48
B. F. W.	27	Cirrhosis of liver, early	101	94	66	64	1.53	1.47	4.65	3.91	4.62	3.20
K. B. E.	23	Anxiety	65	62	112	105	0.58	0.59	6.27	6.38	13.44	10.50
E. S.	34	Penicillin reaction	80	78	64	79	1.25	0.99	4.15	3.97	4.48	6.32
T. D. L.	28	Benign pericarditis	80	78	54	54	1.48	1.44	3.47	3.83	5.40	4.32
P. E. G.	41	Anxiety	86	89	67	112	1.28	0.79	3.35	7.57	5.36	14.56
W. F. B.	19	Diabetes mellitus, controlled			57	94			3.01	4.77	9.12	1.88
Mean	33		83	82	67	73	1.28	1.20	4.07	4.36	6.51	5.92
P Value				>.80		>.30		>.30		>.50		>.60

* Control.
† Experimental.

and CMRglu are not significantly different from the pre-treatment control values for these functions. The oxygen, CO_2 and glucose contents, pH, and PCO_2 of arterial and cerebral venous blood, arterio-cerebral venous oxygen and glucose differences, and arterial oxygen extraction ratio are likewise unaltered by the oral administration of Benzedrine sulfate.

On 4 occasions, 2 in the control period and 2 in the experimental period, exceedingly high values were obtained in the calculation of CMR_{O_2} ($CMR_{O_2} = CBF \times \frac{A\text{-}VO_2}{100}$). We believe this to be a consequence of technical errors made in the determination of CBF in these 4 subjects. In each instance the CBF was unusually high (88, 112, 105, 112 cc/min/100 gm.), while arterio-cerebral venous oxygen difference remained well within the normal range.

The subjective and objective manifestations of the drug were carefully observed and recorded by one of us (J. G. A.) during the experimental period in 11 of the 13 subjects (table 3).

DISCUSSION

The results of this study, which demonstrate that the oral administration of Benzedrine sulfate does not affect CBF, CMR_{O_2} or CMRglu in man, are in agreement with findings of Abreu *et al.* (1). Statistical analysis of their data reveals that significant changes in mean CBF ($P = >.05$) and CMR_{O_2} ($P = >.30$) did not occur when the drug was administered intravenously. The principal difference in the findings of these two studies lies in the fact that the increase in MABP that occurred after the intravenous administration of Benzedrine did not occur when the drug was taken orally. Psychologic manifestations were common. Anorexia was a prominent subjective complaint in 73 per cent of the 11 subjects; insomnia in 54 per cent; increased nervous tension in 45 per cent; and euphoria in 36 per cent. Two (W. F. B. and O. W. W.) exhibited no apparent subjective or objective response to the drug, and one (C. L. N.) became depressed and suspicious during the experimental

TABLE 2.—BLOOD CONSTITUENTS BEFORE AND AFTER THE ORAL ADMINISTRATION OF BENZEDRINE SULFATE

Subject	Arterial Blood												Venous Blood											
	Oxygen content, vol. %		CO_2 content, vol. %		Glucose, mg. %		A-VO_2, vol. %		Oxygen extraction ratio (ERO_2), %		A-V glu, mg. %		Ph		PCO_2, mg. Hg		Oxygen content, vol. %		CO_2 content, vol. %		Glucose, mg. %		Ph		PCO_2, mm. Hg	
	C*	E**	C	E	C	E	C	E	C	E	C	E	C	E	C	E	C	E	C	E	C	E	C	E	C	E
W. C. P.	19.95	19.81	44.54	44.88	64	65	6.52	6.21	33	31	6	12	7.36	7.33	35	38	13.43	13.60	49.65	51.55	58	53	7.31	7.26	43	52
W. L. M.	18.53	16.95	44.38	45.47			6.32	5.95	34	35	11		7.34	7.34	37	37	12.21	11.00	49.76	51.54			7.30	7.30	45	47
O. W. W.	16.05	15.96	47.73	46.59			6.15	6.18	38	39			7.32	7.32	41	40	9.90	9.78	53.94	53.25			7.26	7.27	54	53
J. M. L.	12.27	12.28	48.77	48.04	63	68	5.77	5.69	47	46	9	7	7.34	7.38	44	37	6.50	6.59	54.98	53.48	54	61	7.30	7.33	47	46
C. L. N.	18.53	18.39	44.88	44.70	82	76	7.50	6.58	40	36	9	8	7.36	7.35	35	34	11.03	11.81	51.34	51.17	73	68	7.32	7.28	45	52
K. E. H.	16.91	16.43	48.94	47.34	74	83	4.58	4.28	27	26	8	1					12.33	12.15	53.30	52.32	66	82				
J. L. P.	17.12	17.71	45.39	44.47	85	84	6.59	6.93	38	39	13	12	7.30	7.42	40	31	10.53	10.78	50.86	51.23	72	72	7.28	7.35	49	42
B. F. W.	20.50	17.55	45.61	46.77	67	62	7.05	6.11	34	35	7	5	7.44	7.45	38	37	13.45	11.44	52.01	52.57	60	57				
K. B. E.	18.18	18.16	56.23	53.76	63	61	5.60	6.08	31	33	12	10	7.48	7.39	40	45	12.58	12.08	60.65	58.78	51	51				
E. S.	16.83	15.17	46.95	48.34	84	85	6.48	5.03	39	33	7	8	7.40	7.46	39	36	10.35	10.14	52.66	54.10	77	77				
T. D. L.	19.87	19.52	45.43	43.05	79	80	6.42	7.10	32	36	10	8	7.43	7.44	36	34	13.45	12.42	50.99	50.25	69	72				
P. E. G.	16.58	18.70	51.85	51.59	84	75	5.00	6.76	30	36	8	13					11.58	11.94	58.31	58.10	76	62				
W. F. B.	19.24	18.02	48.55	49.08	274	219	5.28	5.07	27	28	16	2	7.32	7.33	49	48	13.96	12.95	53.30	53.62	258	217	7.28	7.28	56	58
Mean	17.74	17.28	47.63	47.24	93	87	6.10	5.94	36	36	9.7	7.8	7.37	7.38	39	38	11.64	11.28	53.67	53.23	83	79	7.29	7.30	48	50
P Value		> .50		> .75		> .75		> .9		> .85		> .3		> .90		> .2		> .70		> .85		> .85		> .90		> .45

* Control.
** Experimental.

TABLE 3.—SUBJECTIVE AND OBJECTIVE MANIFESTATIONS OBSERVED DURING THE ORAL ADMINISTRATION OF BENZEDRINE SULFATE

Subject	Feeling of Well Being	Anorexia	Insomnia	Increased Nervousness	Malaise	Euphoria	Depression	Paranoid Reaction
W. C. P.		x	x	x	x			
W. L. M.		x	x	x				
J. M. L.	x	x				x		
C. L. N.		x	x	x			x	x
J. L. P.	x	x	x	x		x		
K. B. E.		x						
E. S.			x					
T. D. L.		x						
P. E. G.		x	x	x	x	x		
W. F. B.	None							
O. W. W.	None							
Total	2	8	6	5	2	3	1	1

period. There was no apparent relationship between these manifestations and the cerebral circulatory or metabolic alterations that were observed in the individual subjects. The psychological changes noted in subjects W. C. P. and P. E. G. were identical although the cerebral circulatory and metabolic changes that occurred in these 2 subjects mark the extremes, in opposite directions, of the study.

CONCLUSIONS

1. Cerebral circulatory and metabolic functions have been determined before and after the oral administration of Benzedrine sulfate in 13 male subjects.

2. The oral administration of Benzedrine sulfate has no effect upon cerebral blood flow, cerebral vascular resistance, cerebral oxygen or glucose utilization in man.

3. The psychologic manifestations of the oral administration of Benzedrine sulfate are not related to changes in cerebral blood flow or to cerebral oxygen or glucose consumption.

This work was done with the technical assistance of Mrs. Angeline M. Hayes and Miss Mildred T. Brennan. Mr. Tyree Loving and Mr. W. R. Hampton also gave valuable assistance.

BIBLIOGRAPHY

1. Abreu, B. E., Liddle, G. W., Burks, A. L., Sutherland, V., Elliott, H. W., Simon, A., and Margolis, L.: Influence of amphetamine sulfate on cerebral metabolism and blood flow in man. *J. Am. Pharm. A., 38:* 186, 1949.
2. Bradley, C., and Bowen, M.: Amphetamine and school performance. *Am. J. Orthopsychiat., 10:* 782, 1940.
3. Cutler, M., Little, J. W., and Strauss, A. A.: The effect of Benzedrine on mentally deficient children. *Am. J. Ment. Def., 45:* 59, 1940.
4. Dumke, P. R., and Schmidt, C. F.: Quantitative measurements of cerebral blood flow in the Macacque monkey. *Am. J. Physiol., 138:* 421, 1943.
5. Kety, S. S., and Schmidt, C. F.: The nitrous oxide method for the determination of cerebral blood flow in man: Theory, procedure and normal values. *J. Clin. Invest., 27:* 476, 1948.
6. Livingston, S., Kajdi, L., and Bridge, E. M.: The use of Benzedrine and Dexedrine sulfate in the treatment of epilepsy. *J. Pediat., 32:* 490, 1948.
7. Mann, P. J. G., and Quastel, J. H.: Benzedrine (β-Phenylesopropylamine) and brain metabolism. *Biochem. J., 34:* 414, 1940.
8. Molitch, M., and Eccles, A. K.: The effect of Benzedrine sulfate on the intelligence scores of children. *Am. J. Psychiat., 94:* 587, 1937.
9. Nelson, N.: A photometric adaptation of the Somogyi method for the determination of glucose. *J. Biol. Chem., 153:* 375, 1944.
10. Peoples, S. A., and Guttman, E.: Hypertension produced with Benzedrine. Lancet, *1:* 1107, 1936.
11. Sargant, W., and Blackburn, J. M.: The effect of Benzedrine on intelligence scores. *Lancet, 231:* 1385, 1936.
12. Scheinberg, P., and Stead, E. A., Jr.: The cerebral blood flow in male subjects as measured by the nitrous oxide technique. Normal values for blood flow, oxygen utilization, glucose utilization and peripheral resistance, with observations on the effect of tilting and anxiety. *J. Clin. Invest., 28:* 1163, 1949.
13. Schmidt, C. F., Kety, S. S., and Pennes, H. H.: The gaseous metabolism of the brain of the monkey. *Am. J. Physiol., 143:* 33, 1945.
14. Singer, R. B., and Hastings, A. B.: An improved clinical method for the estimation of disturbances of the acid-base balance of human blood. *Medicine, 27:* 223, 1948.

THE HERMAN HOSPITAL
HOUSTON, TEXAS

THE GERM WARFARE STATEMENTS: A SYNTHESIS OF A METHOD FOR THE EXTORTION OF FALSE CONFESSIONS

GEORGE WINOKUR, M.D.

Introduction

Since man has fought man, attempts have been made to obtain military information by even the most vigorous methods of torture. This is often sporadic and not infrequently does not come before the public eye. In the last several years, however, the tenor of interrogation has changed to such an extent that the emphasis has been placed on more subtle, less grossly physical methods. A successful attempt was made by the Chinese Communists to extort false confessions from men in the Armed Forces of the U. S. who were incarcerated in Korean POW camps, and in the process no resort was made by the captors to the latter-day successors of the rack and the screw. This paper is concerned with the mechanism whereby these false confessions were obtained. Three subjects who signed false statements that they had participated in germ warfare against the enemy of the United Nations in the Korean conflict were interviewed. Material was gleaned from them that was then utilized in the formulation of a synthesis of a method for the extortion of false statements. This paper then is concerned with the presentation of the salient facts of the interviews and the synthesis derived from these.

Interviews

The first interviewee was Mr. A., who was shot down in the summer of 1952 while on a mission to North Korea. He was taken prisoner by the North Koreans and shortly thereafter was delivered to the Chinese and taken to a collecting point where he was separated from his crew and interrogated. He was asked at this time whether he had participated in bacteriological warfare. In conjunction with the interrogation, food became less and worse, and he was made to sit at attention for long periods of time. The Chinese began to press Mr. A. to write a germ warfare confession and at the same time tried to get him to write anything true or false. They would insist that the air speed of a particular airplane, a fact which they had, was faster than it was in reality and would be "tickled" if in exasperation Mr. A. would agree to a faster but false air speed. He was never touched physically in any severe fashion. Mr. A. had stopped smoking prior to his going overseas, but the Chinese insisted that he smoke. Four or five months after his capture they deprived him of cigarettes and offered them as a reward for him to say things. Mr. A. said in the interview that the Chinese became most angry when he insisted that something was true, that was indeed true. In the middle of July the Chinese were infuriated with his poor cooperation and put him before a military court martial where he was told he participated in germ warfare. He denied this vigorously and refused to sign or write anything about it. At the time of the court martial he had a feeling in his stomach of being hungry but points out that he did not desire food. He was convicted as a war criminal and sentenced to be shot at a later date.

Mr. A. was taken to another camp and placed in a cave where he lived in semi-darkness with 14 ROK prisoners. The area was small, and the prisoners were packed in. One of the ROK prisoners was in handcuffs, and he was taken out of the cave. A volley of shots was heard by Mr. A., and the Chinese walked by the cave with a coffin-like box and the handcuffs. Lice were rampant; the cave was very hot; the rain came in and darkness prevailed. Food was filthy, and he had welts all over his body. Mr. A. was told by his captors that his crew members had confessed to germ warfare. He continued his refusal to comply with their demands for a confession. On one occasion he was brought out of the cave, and eight or ten POW's, members of a B-29 crew, were shot in his sight (during the interview, Mr. A. became tearful at relating this). The Chinese told him they had had him witness this purposely, and that if he did not confess, the same thing would happen to him. They dwelled on the idea that

he was inhuman and a criminal to all humanity and tried to make him feel guilty. Mr. A. insists he felt no guilt, only fear. His crew and family at home were threatened. He describes his condition at that time in these words: "blank, physically beat"; "couldn't put anything together"; "too many things came to my mind"; "didn't know what to think about." Mr. A. prayed and fingered a rosary which had been given to him by a friend, but at that time the idea came to him that God had no control over the Chinese although He did have control over other people; consequently, he dropped religion. The idea of returning the rosary to his engineer, who had given it to him, became paramount. Mr. A. had a lot of time to himself, and the execution weighed heavily on his mind. He started to wonder what the Chinese were after and felt that something would have to give and wondered what would happen if he signed a confession. He realized that a confession would be a lie, and he could not remember from his briefings if it would be wrong to tell the enemy a lie. Mr. A. pointed out that soon he did not know whether it was right or wrong to sign a confession. He just wanted to get the pressure off and wanted to get back to the States and tell the people what the situation was. He felt this was more important than dying in Korea. Further, he pointed out in the interview that "lies were what they were after" and that the "Chinese eat lies up." However, he reiterated that he had nothing to say or sign, and he was taken out of the cave, given a bath and DDT and was returned to another camp and put in another cave. There the situation was almost as bad. After continuous refusals he was taken out of the cave, given a haircut and shave and was sent to another camp. At this other camp, he was put in a Korean house and lived 24 hours with a Chinese interrogator who at first tried to be friendly with him. Mr. A. was asked for military information and either refused or lied in response to this. Interrogations became extremely long and would last at this time from 6:00 a.m. to 1:00 or 2:00 a.m. the next day. There were one to two meals a day. The quantity of food was small. The interrogator, however, was eating well in front of him. Mr. A. knew they had the information that they wanted from him, but he would not admit it. His captors would give him false answers and ask him to write these answers down. He would write down the false answers in order to get the pressure off. The Chinese were satisfied only when the answers about his personal life were completely false. For instance, they asked him the occupation of his father, and he told them that his father was a farmer. The captors insisted that he was a factory worker, and Mr. A. agreed. He was asked about his mother and said she was a housewife. His captors insisted and he agreed that she, too, was a factory worker. They insisted that he was not a high school graduate, and he agreed although he had gone to college. The interrogator was quite hostile and attempted to get him to sign bacteriological warfare confessions. Mr. A. collected lead toothpaste tubes at this time. Apparently in the POW camp he hoarded a great deal. He wanted to kill his interrogator and consequently ground up the lead tubes. When the interrogator's back was turned, Mr. A. dropped the lead into the interrogator's food. The interrogator ate it, became ill, and never returned. There were several more court martials. Frequently Mr. A. was asked to sign germ warfare confessions. The weather was freezing, and he had no heat. He was unable to move his bowels for days on end. He frequently was not allowed to urinate, but he was never incontinent. He would often sneak to a corner of the room and urinate. He was offered clothing as bait and also tobacco if he would sign the germ warfare confessions. Around the end of Autumn, 1952, he was given a meal, warm clothing, and tobacco and was told that he would have good treatment if he confessed. Up to this time for six days he had had no food. In December, 1952, Mr. A. was taken to another camp, and there it was pointed out by his captors that other prisoners had a fairly good time of it. A new interrogator, who acted somewhat pleasantly toward him, arrived. On Christmas Day he was given a good meal and a Christmas card and some candy. The next day he was taken to a radio equipment shack and heard over radio Peiping that he had confessed to bacteriological warfare. He was court martialled again, this time based completely on bacteriological warfare. At that time the Chinese told him that he should live and help his country and do what

he believed in. Early January, 1953, was extremely cold. Mr. A. started to think that he ought to live because he had the idea that he ought to do something, that he had been put on earth for a definite goal. He felt that his own country would not harm him for telling a lie, but he still thought possibly he might be doing wrong in aiding the enemy. He felt perhaps if he gave something fantastic to the enemy, it would be all right. He felt that if he did not give them anything at all in the way of a confession, he would not return. His weight had gone down from 170 pounds to about 100 pounds. He had weighed himself with a makeshift scale during some period of time in Korea. In February, 1953, he wrote out a fantastic story in accordance with an outline that was given to him. Conditions had been getting worse. He had been forced to cut wood but was not allowed to burn it. The Chinese were dissatisfied with his written confession and brought it back for further work. Mr. A. was so angry he tore it up and threw it away, which so infuriated his captors that he again went 6 days without food. His memory had become poor, nothing seemed logical to him, nothing connected. Insignificant things were prime factors. His body he feels at that time was a solid scab from the waist down. He felt that the Chinese would get military information from him if they kept on, and he did not under any circumstances want to give them that. He was brought something to sign and did not know exactly what it said, but he signed it in a printed manner rather than by writing, feeling that this was one small way of keeping his signature from being on a confession. Thirty minutes after he printed his name under the confession, he was given fire, insecticide, cigarettes and food. Conditions improved. He was moved to another camp, and conditions improved some more. Interrogations at this point became nil. He was given a great deal of Communist literature, and it was attempted to get him to write book reports. However, he refused. The Communists did not press him. He frequently argued with his captors over various points, particularly points in the Communist manifesto, and he constantly threw the idea in their faces that it was necessary that they adhere to the Party Line and not think independently. In the latter part of April, 1953, Mr. A. was joined by another American prisoner. Up to the time the other prisoner came he had spent 330 days in essentially solitary confinement. He was once more moved to another camp in June, 1953. He was gaining weight, food was fairly good, there was no more interrogation. He heard about the war being over and was given a statement about bacteriological warfare to sign. If he did not sign this, his captors threatened him with the idea that he would not be leaving to return to his own country. He signed the statement once more in a printed form. He made a tape recording which he read from a piece of paper. He was given his choice to stay with the Chinese or be repatriated and chose the latter. He was consequently returned to the United States.

Mr. A. at the present time feels that it is necessary that he tell his compatriots about conditions under the Communists. He feels good in his knowledge of what can and will happen if things are left to continue as they have been going. He feels he has a deeper sense of responsibility and has now more of a reason to live. He further feels he has a better understanding of life and people.

The second interviewee was Mr. B. Mr. B. at the time of the interviews with the author in late June, 1954, had pain in the right scapular region with torticollis. This became worse when the patient was under some sort of emotional strain. Further, he had an unusual tic which consisted of his frequently clacking his teeth. Mr. B. bailed out of an airplane in the summer of 1952. At this time he was stuck in the hatch and passed out and was jettisoned by the plane. His neck, right shoulder and arm were in the slipstream, and his body was bent. He was taken captive by the North Koreans and had heard that the North Koreans were extremely hostile toward American airmen. He was delivered into the hands of the Chinese where similar experiences occurred to him as happened to Mr. A. The Chinese would insist on wrong answers, for instance, the number of bombs on a wing. They insisted that Mr. B. say there were a certain number of bombs on a plane instead of the right number, which they knew perfectly well. He was told by his captors that they knew he helped in bacteriological warfare, and that they wanted a confession

from him. Food at that time became insufficient, housing was in small and inadequate quarters. Mr. B. had a difficult time trying to get to the latrine. On one occasion he had to defecate in his pants because they would not let him go to the latrine, and once he was almost shot when he tried to take a "leak." This treatment alternated with periods when the Chinese would be rather nice toward the prisoner, and he was never sure from one day to the other what would happen. If he refused to confess, they told him he would not be repatriated. Five weeks after being taken captive, he was brought up for military court martial and was told that the rest of his crew had confessed, which was untrue, a fact which he could not know. He was offered the alternatives of rewards for signing statements and punishments for refusing to do this. At this time Mr. B. felt he would be killed. Interrogation continued with constant threats of death and violence. There was poor sanitation. Mr. B. defecated in his pants one to two times. On three or four occasions he was brought before high ranking Chinese officers who brought Mr. B. a confession. Early in August, 1952, Mr. B. told the Chinese that it was possible that germ warfare was occurring but that he knew nothing about it. He was then moved up north and came across Mr. A. Mr. A. told Mr. B. of having seen a Korean in chains being killed and about the shooting of the B-29 crew. Mr. B. was also certain the Communists would carry out their threats. On arriving at his new camp, Mr. B. was put in solitary confinement in a Korean hut and interrogated on military information. He was questioned on things he knew nothing about and finally would tell the Chinese the answers they wanted him to give. On one occasion he was asked to make a diagram of a piece of apparatus. Mr. B. has no knowledge of this apparatus, as it was only known to the pilot, but he drew for them an imaginary device with which they were quite pleased. In October the germ warfare statements and questions started again, and about this time Mr. B. produced an untruth; namely, that he flew a mission in China and that it might have been for the purposes of germ warfare. There was a great lack of food at this time. He was kept on a one-legged stool for 12 hours a day for six or seven days. There was the same insistence on the part of the Chinese for false answers, and Mr. B. points out that "It was easier to give what they wanted. We weren't giving any information." In late December Mr. B. was removed to another camp. At this time he could hear Christmas carols in the background, started to cry, lost all interest, became quite despondent and thought of killing himself. It would seem that he was probably retarded in psychomotor activity at this time. "I just stopped thinking." "The world didn't mean too much to me, it was 40° below zero, there was no fire, no heat." Mr. B. called himself at this time a "numb animal." He thinks that his weight went from 145 pounds to somewhere around 100 pounds. He was constantly thirsty because the water was frozen. He was brought a statement to read and sign and was taken to a warm room in a headquarters building and was given the paper to sign. At the first part of the new year Mr. B. signed his first germ warfare statement and then made a tape recording in a sound movie falsely admitting germ warfare and having bombed China. Finally after 9½ months of essentially solitary confinement, another prisoner was brought to live with him. From then on there was relatively good treatment, and Mr. B. was allowed to have daylight and was able to talk. Some attempt was made to indoctrinate Mr. B. with Communist literature, but because of his continuing arguments he was consequently left alone. In the autumn of 1953 germ warfare confessions and recordings were redone and backdated. He was told at this time that if he did not rewrite this material, he would not be repatriated. This having been done the patient was sent back to the United States.

At the present time Mr. B. feels that he has done the best that he could. He feels he has followed intelligence briefings which had not dealt specifically with what to do in situations of this sort and had to shift for himself when finally taken captive. He did not at any time give military information and does not think he has done anything wrong. A fine point comes up with Mr. B. as it has with Mr. A.—namely, is it right or wrong to tell lies to the enemy.

The third interviewee was Mr. C. who was shot down over Korea in the winter of 1951. He was shortly taken to a MIG base where he

was questioned about military information, threatened with "death at dawn" in a rather dramatic manner and threatened by having a gun placed on the table, cocked and waved in his face. He refused to answer questions and facetiously told his captors to get the book which told the answers to most of the things they wanted to know. They proceeded to produce the book for Mr. C., and thereafter when they asked him questions, he would answer them from the book, feeling that they already had the answers and he was contributing nothing to them. He did at that time lie about various facts; namely, the number of planes in his group and about his commander. After nine days of interrogation of a rather intensive nature, he was taken to a prison camp where a Korean colonel came in and in an angry fashion told Mr. C. that everything he had produced was lies. Mr. C. went through the same material, namely these lies which he had memorized, and these were accepted again on this occasion. After being moved a couple of more times, he was taken to a collecting point where he was plied with questionnaires. These were filled out in such a manner as Mr. C. felt he gave truthful information only when he was sure that his captors already had this information. The rest of the material he gave was essentially a lie. At that point he did not feel that lying was "cricket," and at the time of the interview with the author he had an abhorrence about lies. Of the filling out of the questionnaires he says, "you could bullshit them as long as you wrote." He was brought together with other prisoners two weeks after capture. His leg, which had been injured prior to his capture was painful, and he was forced to walk about this time a great deal. Food was poor, consisting mostly of seaweed, soup and rice. By the end of February Mr. C. had difficulty with his throat which was inflamed. He was interrogated 5 or 6 hours a day and still was producing lies on questionnaires. He and the other prisoners were able to produce consistent lies because they had agreed that they would not answer any questions which did not pertain to their own field of endeavor. Early in March the prisoners were asked if they would broadcast over Chinese radio. They said they might do this, feeling if they could get their names known to the United Nations forces, it would be worthwhile agreeing to broadcast. It was around this time also that they were immunized against various bacteria that the Chinese said were used by the United Nations in germ warfare. Mr. C. was put to work making a history of the F-86 in Korea, and this lasted about a month. It was a complete fabrication. About the first of April Mr. C. was unable to eat anything except rice. His leg was quite painful, but he was being forced to carry heavy quantities of wood. By the 10th of May many more men were brought in, and forty people were crowded into a very small space. At this time Mr. C. and his cohorts were planning to escape and were waiting for rain. Before this could be done, he was moved to another camp, and after a series of changes he wound up at Camp #2, where he was put in "isolation" with a Chinese interrogator who spoke English. This was 1 June, 1952. He was interrogated from dawn until dark. Diarrhea manifested itself, and Mr. C. found that when the interrogation was getting "rough," he could get out of the interrogation by running to the toilet to have bowel movements. There was constant pressure. He would sit on a chair for long periods of time, was threatened with his life and was having great frequency of urination and 11 to 14 bowel movements per day. At one point in his captivity he had a prolapse of the rectum. He was getting very little to eat. He was suffering from a toothache, and around June 6 or 7 he was once more given immunizations. At this time he started to say the Lord's Prayer over and over again. He was unable to sleep. He would become angry with his interrogator and lost his temper when the interrogator had attacked God, and at this time he begged the interrogator to kill him. On occasion he would say to the interrogator, "What do you want me to tell you—a lie?" He became irritable, cried frequently, and suffered from tinnitus. He had feelings of guilt because he did not know how long he could go on refusing to answer the questions. He made up a story because he thought it would be better to make up a story than to sign the lies they produced for him. The story was so wild and fantastic that they were dissatisfied. He handed in a second article, and at this time he became ill with fever, chills and chest cramps. He lost consciousness. He came out of the coma on July 4.

By 10 July he was moved, but this time he was kept from going to the toilet and was constantly harrassed and given orders. His prior statements were not considered any good because they were considered too tentative, and he started to make up a story in his head. By July 26 or 27 he had confessed falsely to dropping bacteria over Communist territory, and he lied about other people on the mission. He was at this point feeling as though he were guilty of treason, but he did feel as though he held out as long as possible. He was extremely tense, was unable to talk, and if a stranger would come into the room, he would start to shake. This shaking continued until around January 1953. He finally recorded a confession for his captors, and also around this time he was given an article which the Chinese said he had written, but which Mr. C. did not remember. He assumed he had written it at the time he became ill prior to July 4, just after the Chinese had refused his first two statements. He was given vast quantities to read which were essentially Communist in their orientation. During this time he was living with a Chinese interrogator who was called a "comrade." Mr. C. at this time was extremely irritable toward the Comrade. One day he became angry when ordered to brush his teeth. He refused, and at this point the Comrade left. By the last of October he had left for good. By that time Mr. C. was sharpening up a piece of metal with which he was going to kill the Comrade. After the Comrade left Mr. C. said he had the "thousand-mile stare." He did not eat everything he was given, he was apathetic, he dreamed a great deal of his wife, felt that he was with her and having intercourse with her. He would walk outside his hut in a dissociated manner and then come to the realization that he was still in Korea. This ended around Thanksgiving Day, 1952. Mr. C. signed more statements and made more false identifications of people who were alleged by the Chinese to have been in germ warfare raids. Around Christmas he moved in with another POW who was also one of the people who had been severely interrogated as far as the germ warfare situation was concerned. Around February, 1953, Mr. C. was arguing so vigorously with his captors about Communist tenets that some of the books on Marxist economics were taken away from him. On August 15 he rewrote his old statement. The last statement Mr. C. signed had dates in it which he presumed would invalidate the statement entirely. Mr. C. was repatriated September 1, 1953.

Mr. C. seems to have suffered during the time he was in the POW camps from a gross stress reaction with severe anxiety and dissociative episodes. History taken from Mr. C. as regards his social and genetic development indicates that he was a well-adjusted young man and showed no signs of neurosis or psychosis prior to his being in the POW camps.

Discussion

A word should be said about the present circumstances of the above subjects. In none of the cases were they seen because of their needing psychiatric help. All of them were functioning at the time seen at an adequate social and work level.

The monotonous repetition of the various stresses suffered by all three of the subjects is considered necessary for one reason: namely, to rule out the idea that these men broke down because of chronic pre-service impairment of personality. All of these men were interviewed for about four hours each by the author, and from the past histories there was no reason to append psychiatric diagnoses of neurosis or severe character disorders on any of them. Mr. C. had a complete psychiatric work-up with psychological tests which showed him to be free from mental disease or psychopathic disorder. The entire complement of Mr. A's group was interrogated and apparently signed false germ warfare statements. It seems highly unlikely that the Chinese considered *all* of them weak individuals and likely to break under stress. Thus, although there is some work on the idea that emotional background predisposes to doing the bidding of the authoritative figure, it is thought here that the extreme severity of the stress obviates the usefulness of this concept in these cases (4). Additional proof of this is that these men broke only in a particular sphere, i.e., that surrounding germ warfare. Attempts to indoctrinate them with Communist theory met with resistance on their parts which in turn caused a discontinuance of the effort on the part of the Chinese.

The reason for the breakdown in the one particular sphere will be dealt with below.

The mechanics of the processes involved in the false confessions are capable of being expounded with the use of the various facts that are known. The frame of reference used will be that of learning theory as discussed by Dollard and Miller (1), in which they point out that a great deal of learning takes place by the reduction of drive. The drive state of the men who signed false confessions was produced and intensified by conflict which, experimentally and clinically, has been proven to build tension (3). The conflict in the states alluded to above consisted of the uncertainty as to status in captivity, the inconsistencies of the captors in one day dealing fairly with the men and the next day harshly. The threat of severe punishment and death provided further conflict as there were no ways of successfully coping with the onslaught of the desires of the Communists. The conflict was between the possibility of death and the possibility of life with marked guilt feelings. Further the question of starvation provided a similar conflict as above. Mr. A. lost about 40 per cent body weight and Mr. B. about 33 per cent body weight. Research done on food deprivation has indicated that a 24 per cent loss of body weight resulted in pronounced depression, irritability and subjective feelings of impairment of judgment and comprehension (2). All of these symptoms would combine to elevate the drive state. Deprivation of heat and adequate clothing and the deprivation of tobacco after habituation had been forced on the men also were factors in the production and maintenance of a high drive state.

The possible solutions to this situation for these men were threefold: acquiescing to the demands of the captor, death from physical debilitation, or intense anxiety with the possibility of psychosis. However, it is to be noted here that in one way the histories of all three subjects were similar; namely, the reduction of the hostility of the enemy and his satisfaction when the subjects produced lies. The Chinese had these POW's write lie after lie on paper and had these men tell what seemed to them nonsensical falsehoods; and in response to this the captors reduced the pressure of their incessant interrogation. Thus it becomes clear that the men involved were trained to lie in order to protect themselves against the intolerable conflict with their captors which engendered drive. Some proof of this idea is contained in a statement of Mr. C. who remarked in a deposition written *before any of the subjects had been interviewed by the author of this paper*, "Also, it is very difficult for me to both tell the truth and give answers at all when being questioned seriously by anyone."

The question arises as to why more than one statement was made by these men. Behavior is learned by drive reduction, and the successful coping with the problem resulting in relief from tension made it much easier to comply with the later demands by the Chinese. Thus the repeated statements, broadcasts, etc., were nothing more than the repetition of a learned sequence of behavior. The guilt that was aroused by the making of the statements was handled by the mechanism of rationalization in which the subjects questioned whether telling lies to the enemy could be harmful to their own country. Further, starvation and illness may have decreased their perception to such an extent that they could not adequately make this evaluation. In addition, the idea that they had to return in order to provide information to their own country about the methods of the enemy may also be evidence of rationalization.

Summary

This paper deals with the material gleaned from three subjects who falsely made germ warfare confessions in Korea. The stresses to which they were subjected are presented, and it is postulated that these men were trained to lie by the Chinese as a protection against intolerable conflict and stress. The guilt that was engendered by these confessions was handled in the individual by the mechanism of rationalization.

Addendum

Collateral evidence for the above synthesis was brought to light in an article appearing in the October 21, 1954 issue of the St. Louis Post Dispatch. An Army corporal was badgered by his captors in Korea to "confess" and finally confessed to two things (difficulties in the POW camp) neither of which was related to him. The corporal remarked that his inter-

rogator finally felt he was getting somewhere, but that he was not completely satisfied. An accidental physical disability produced much-wanted respite for the prisoner by getting him out of the situation.

BIBLIOGRAPHY

1. Dollard, J., and Miller, N.: *Personality and Psychotherapy*. New York: McGraw-Hill, 1950.
2. Franklin, J. C. et al.: Observations of human behavior in experimental semi-starvation and rehabilitation. *J. Clin. Psychol., 4:* 28, 1948.
3. Gantt, W. H.: Experimental Basis for Neurotic Behavior. New York: P. B. Hoeber, 1944.
4. Redlich, F. C. et al.: Narcoanalysis and truth. *Am. J. Psychiat., 107:* 586, 1951.

4580 Scott Ave.
Saint Louis, Missouri

AN ANALYSIS OF REGIONAL DIFFERENCES IN MENTAL ILLNESS[1]

CAROL BUCK, M.D., PH.D., D.P.H., J. M. WANKLIN, B.A., AND G. E. HOBBS, M.D., M.P.H.

A. PURPOSE

It has repeatedly been shown that first admission rates to mental hospitals vary (a) between geographic areas, within parts of one country and even within zones of one city (2, 3, 7, 9) and (b) with certain demographic and social variables such as urban-rural residence, marital status, native or foreign birth, economic status and amount of education (4, 6, 10, 12, 13).

Obviously, the observed geographic and regional variations in incidence might arise from the fact that areas differ in respect to the population variables described above. The purpose of the present study is to determine to what degree regional variations can be explained in this manner.

B. METHOD

1. Regions studied

The regions studied were the 14 counties of Western Ontario whose combined population is about a million (approximately 20 per cent of the total population of the Province). The southern counties of Western Ontario include densely populated industrial areas and rich farm land, while the northern counties are characterized by more sparsely settled and rather unproductive areas. Two counties have cities with populations exceeding 100,000, one being a university center and the other a highly industrialized cosmopolitan city. Thus, although the area is not extensive, the diversified characteristics of its counties are highly suited to an investigation of this kind. Furthermore, first admission rates for the period under study have already been analyzed for the area as a whole in terms of rural-urban residence, marital status, nativity and education (15).

2. The sample

The study sample consisted of the 1,983 first admissions from Western Ontario who, between January 1, 1950, and December 31, 1952, entered either of the two Provincial mental hospitals[2] serving the area. Both hospitals are within convenient distance of the University.

Admissions from the 14 counties to other more distant hospitals in the Province were excluded because the detailed nature of the study required intimate review of case histories and access to the patients.

Therefore patients excluded from the study were 1) juvenile mental defectives, epileptic and tuberculous mental patients and the criminally insane who are sent to specialized Provincial institutions, and 2) patients admitted to a Federal Veterans' hospital and to a small private sanatorium. Thus, most juvenile patients and a disproportionate number of adult males were eliminated from the sample.

Fortunately, no regional bias which could have distorted the results of the present analysis was introduced through these omissions. On the basis of a tabulation of all Western Ontario first admissions for 1950, published in the Annual Report on Mental Institutions (11), it was established that our sample for that year was representative of the total admissions in terms of its distribution by county of residence.

3. The analysis

The analysis involved the correlation of the rank order of the counties on the basis of their first admission rates with their rank order on each of a series of demographic indices obtained from the 1951 Census of Canada. County first admission rates were expressed as the average of the three yearly rates for the period 1950 to 1952, using the 1951 Census population as a base. All rates were expressed per 100,000 population. Spearman's Rank Difference

[1] From the Faculty of Medicine, The University of Western Ontario Medical School, London, Canada. This study was made possible by a Mental Health Grant from the Department of National Health and Welfare and the Ontario Department of Health.

[2] Ontario Hospitals at London and St. Thomas.

method was used to derive the correlations (16).

C. Results

1. Distribution of county rates, ages 15 and over

County rates for the population 15 years and over ranged from a high of 108.8 to a low of 58.4, pointing to a marked variation in the admission level throughout Western Ontario. Furthermore, data assembled from a previous study showed that the range and rank order of the rates had not changed greatly over the decade. A coefficient of +0.66[3] was obtained when the county rates for the period 1950 to 1952 were correlated with those for the period 1940 to 1942.

TABLE 1.—County First Admission Rates per 100,000 Population Aged 15 and Over
Western Ontario, 1950 to 1952

County	Ages 15 & Over		Ages 15–64		Ages 65 & Over	
	Rates	Rank	Rates	Rank	Rates	Rank
Brant	78.6	6	73.4	4	114.0	12
Bruce	65.2	11	47.7	12.5	133.9	9
Elgin	93.0	3	66.9	6	236.6	3
Essex	82.1	5	76.9	3	132.4	11
Huron	58.4	14	50.3	11	96.6	14
Grey	72.3	9	58.6	8	140.7	8
Kent	101.5	2	81.4	2	240.4	2
Lambton	108.8	1	98.6	1	179.8	6
Middlesex	75.0	8	47.7	12.5	245.9	1
Norfolk	84.6	4	70.8	5	185.7	5
Oxford	77.8	7	57.4	9	198.2	4
Perth	59.8	13	43.7	14	141.1	7
Waterloo	69.3	10	63.6	7	113.5	13
Wellington	64.7	12	52.7	10	133.7	10

2. Distribution of county rates by age

Analyses made by ourselves (14) and others (5) had made it clear that admission trends differ with age, suggesting that factors determining the admission level may also have an association with age. In order to explore further this possibility, county rates were kept as age specific as the size of the sample permitted.

A separation of ages 15 to 64 from ages 65 and over was the two-fold grouping adopted, since it was between these groups that greatest differences in the admission trends had already been found. County rates, by age, are shown in table 1. A correlation of +0.06 between the two sets of rates was not statistically significant, again suggesting quantitative or qualitative differences between the factors determining admission levels in each age range.

In figures 1 and 2 the geographic location of counties with high, intermediate and low rates for each of the age groups is shown. There was a general tendency for high rates in both age groups to be clustered throughout the southern regions of Western Ontario. Because of the characteristics of this region, an association of the admission level with the extent of urbanization was at once suggested. A direct comparison of the rates for the rural and urban populations of Western Ontario as a whole, based on this admission sample, had, in fact, already pointed to a marked urban excess (15). It was possible, then, that differences between county rates were simply determined by the rural-urban ratios of their populations.

There was evidence, however, that the relationship was not as simple as this. High county rates at ages 15 to 64 seemed, for instance, to be particularly associated with industrialization, rather than with urbanization *per se*. The three counties having the highest rates—Essex, Kent and Lambton—are outstanding in their industrial activity. On the other hand, a notable feature of the distribution of rates for ages 65 and over was that two of the five counties having the highest admission levels were those in which the mental hospitals were located. For these ages, therefore, the possible influence upon the rates of the distance to a hospital warranted further investigation.

These observations guided the selection of demographic indices which were used for the correlations which follow. However, in many instances, a selection of the most appropriate index was prevented by the limitations of census data.

3. Population indices associated with county rates at ages 15 to 64

(*a*) *General:* County rates at these ages were not directly associated with the rural-urban ratio of their population. The coefficient of correlation between their rates and the percentage of their populations living in urban areas was only +0.18 and was not statistically

[3] Significant at the 0.05 level.

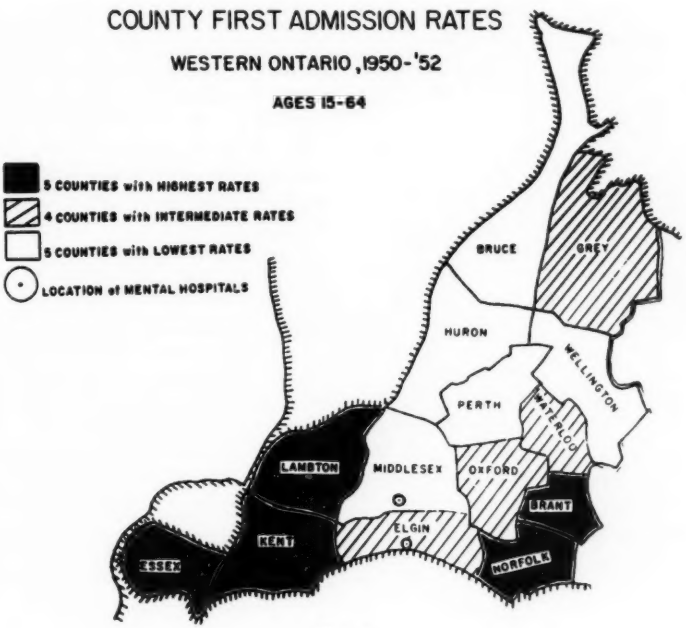

COUNTY FIRST ADMISSION RATES
WESTERN ONTARIO, 1950-'52
AGES 15-64
5 COUNTIES with HIGHEST RATES
4 COUNTIES with INTERMEDIATE RATES
5 COUNTIES with LOWEST RATES
LOCATION of MENTAL HOSPITALS
BRUCE
GREY
HURON
WELLINGTON
PERTH
WATERLOO
LAMBTON
MIDDLESEX
OXFORD
BRANT
ELGIN
KENT
ESSEX
NORFOLK

FIG. 1

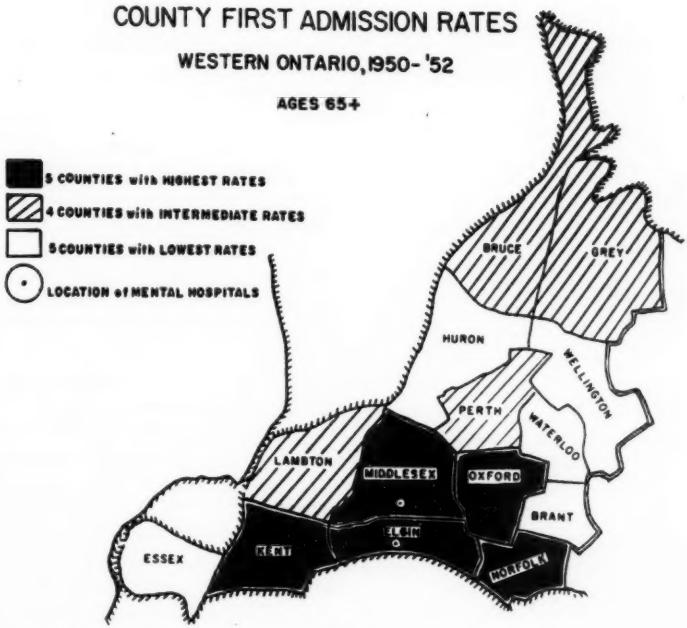

COUNTY FIRST ADMISSION RATES
WESTERN ONTARIO, 1950-'52
AGES 65+
5 COUNTIES with HIGHEST RATES
4 COUNTIES with INTERMEDIATE RATES
5 COUNTIES with LOWEST RATES
LOCATION of MENTAL HOSPITALS
BRUCE
GREY
HURON
WELLINGTON
PERTH
WATERLOO
LAMBTON
MIDDLESEX
OXFORD
BRANT
ELGIN
KENT
ESSEX
NORFOLK

FIG. 2

significant. Nor did the county rates correlate significantly either with the proportion of their populations with 8 years or less of schooling or with the average distance to the mental hospital serving the county.

The rates did correlate positively and significantly, as shown in table 3, column (a), with the following: (a) the proportion of persons under 65 years of age among the population over age 15; (b) the proportion married among the population aged 15 to 64; (c) the median wage of the heads of wage earner families; and (d) the proportion of recent immigrants among the population aged 15 to 64. These indices were, themselves, somewhat interrelated. The multiple correlation of the proportion of the population under 65 years of age with the proportion of recent immigrants and with the median wage was +0.81. As a result, the multiple correlation of the rates with the proportion married and the proportion under age 65 (+0.89) approximated that which would have been obtained by introducing all four variables.

A relatively young, predominantly married, financially prosperous and cosmopolitan population is one usually associated with a thriving industrial area. Thus, although county rates were not influenced by the degree of urbanization, they may have been influenced by the kind of urbanization.

(*b*) ***By population size:*** The first step in the pursuit of the above hypothesis was to establish whether or not the rank order of the county admission rates was determined essentially by the urban segments of the county populations. For the 11 counties having cities with populations of 10,000 or over, rates were calculated separately for these larger urban areas and for the remaining smaller urban and rural areas combined (table 2).

TABLE 2.—COUNTY FIRST ADMISSION RATES PER 100,000 POPULATION BY AGE, URBAN AREAS 10,000 AND OVER AND AREAS UNDER 10,000 POPULATION
Western Ontario, 1950 to 1952

County	Ages 15 to 64				Ages 65 & Over			
	Areas 10,000 & over		Areas under 10,000		Areas 10,000 & over		Areas under 10,000	
	Rates	Rank	Rates	Rank	Rates	Rank	Rates	Rank
Brant	102.7	2	41.3	10	161.0	8	55.1	11
Elgin	86.6	4	57.3	4	340.6	1	179.3	2
Essex	99.3	3	46.3	9	129.9	10	135.9	7
Grey	62.3	9	56.9	5	110.0	11	151.3	5
Kent	85.1	5	78.9	1	310.4	3	214.1	1
Lambton	121.5	1	76.8	2	216.1	5	160.6	4
Middlesex	65.8	7	19.6	11	316.6	2	139.9	6
Oxford	72.1	6	51.7	6	279.0	4	172.0	3
Perth	39.1	11	46.4	8	210.8	6	104.0	9
Waterloo	65.6	8	60.4	3	141.3	9	71.4	10
Wellington	60.1	10	47.3	7	179.4	7	104.5	8

Rates for areas 10,000 and over were generally higher than those for areas under 10,000, but the correlation between the two sets of rates (+0.13) was not statistically significant. Further, the correlation between total county rates and those for areas 10,000 and over was higher (+0.84) than that between the total rates and those for areas of under 10,000 (+0.54). In other words, the rank order of the total county rates was essentially a reflection of the rates for the larger urban areas of the counties.

It was next established that the rates for areas 10,000 and over were the ones which correlated highly with the various population indices described, as may be seen in table 3, columns (b) and (c).

Since data were available for crimes known to the police in the largest city within each of eleven counties (1), it was possible to make some assessment of the relation, if any, between crime and mental disease in these urban areas. Among these 11 cities, the rank correlation between offenses known to the police per 1,000 population and the male rate of first admissions was not significant (−0.08).

One further step was needed before the associations found could be clearly pin-pointed. County rankings by population indices, as by rates, might actually differ between the two types of area (under and over 10,000 population) and when calculated for the counties as a whole, as was done, might be more representative of the larger urban populations. In other words, the possibility had to be explored that the rates for areas with populations under 10,000 might also correlate highly with population indices determined specifically for areas of this size.

Census data permitted the necessary calculation for only one of the indices, the proportion of the population under 65 years of age. The correlation between the rank order of the rates and the specific proportion under 65 in

areas 10,000 and over was +0.59 (approaching significance) and in areas under 10,000 was −0.06. The effect of correlating the rates with the index as it pertained in the appropriate populations was, therefore, one of lowering both coefficients slightly, but the tendency persisted for an association to exist in areas 10,000 and over, and not in those under 10,000 population. One could not be certain that comparable results would have been obtained for the other indices; but at least for the proportion of recent immigrants and for the median wage; which together were related to the proportion under age 65, this seemed likely.

Variations in the first admission rate at ages 15 to 64 among urban areas with populations of 10,000 or over were therefore closely associated with variations in the four demographic variables mentioned, while among smaller urban and rural areas these associations did not seem to pertain.

One explanation for this inconsistency is that the indices may have a different significance in each of the two sizes of population. Among the larger urban areas, for instance, it has already been postulated that a young, predominantly married, economically prosperous and cosmopolitan population is characteristic of the flourishing industrial centers of Western Ontario. Assuming the validity of this postulate, it would obviously not apply to the small urban and rural areas.

Another way of looking at this lack of correlation among the populations outside the larger towns is possible. These populations are geographically more scattered than those of the towns. It may also be that they differ one from another in other ways, so that even if a correlation between a given demographic index and the first admission rate existed within each of them, such correlation might disappear when these heterogeneous populations were grouped together. The fact that a similar phenomenon has been encountered in a rather different sphere suggests that such an explanation is possible. For example, in a Scottish survey of the correlation of stillbirth and neo-natal death rates with post-natal death rates, the correlation coefficient was much higher (+0.53) for towns than for the counties (+0.24)(8).

Even a tentative interpretation of the findings in the larger urban areas was difficult. The correlated indices helped to define the population characteristics associated with variations in the level of admission to mental hospital, but the causative effect, if any, of those characteristics upon the admission level could only be speculated on.

TABLE 3.—RANK CORRELATION COEFFICIENTS BETWEEN COUNTY FIRST ADMISSION RATES, AGES 15 TO 64 AND SELECTEDE DMOGRAPHIC INDICES
Western Ontario, 1950 to 1952

Demographic Index	(a) Total County Rate	(b) Rate for Areas 10,000 +	(c) Rate for Areas Under 10,000
Proportion of population under age 65	+0.73*	+0.70*	+0.30
Proportion of population married	+0.74*	+0.72*	+0.37
Median wage	+0.57*	+0.71*	+0.11
Proportion of recent immigrants	+0.62*	+0.58	+0.11

* Significant at 5% level or better.

Excessively high rates at ages 15 to 64 in areas of industrial prosperity are superficially somewhat incompatible with the results of our previous analysis of the trend in the admission rate in Ontario between the years 1927 and 1946 (14). In that analysis, the admission level was found to decrease with increasing prosperity as measured by the index of employment. This paradox may be more apparent than real, however, for although admissions to mental hospital may decrease during more prosperous times, that part of the population exposed most to the economics of industry may at all times be most vulnerable.

The stressful aspects of industrial urban living are, of course, thought by some investigators to have a detrimental effect upon mental health. Then, too, industrial areas, offering opportunities for transient employment, undoubtedly attract the "drifters", a group within which pre-psychotic personalities are surely more prevalent, and within which the resources for care without hospitalization, in the event of a mental illness, are likely to be few.

Moreover, both a need and a desire for hospital care and treatment of the mentally ill may pervade these urban industrial populations generally. Crowded living conditions could create the need, and successful health educa-

tion may have lessened the social stigma long associated with mental hospitalization.

Finally, the detection of the mentally ill may be particularly certain in such areas. If this is so, it could be the result of at least two factors. First, the public's definition of abnormal behavior may be comparatively severe, and, second, hospitalization may be implemented by the concentration in these areas of physicians whose availability could increase the likelihood of a professional diagnosis and recommendation for treatment being made.

It was thought that if the latter factor were operative, it might be reflected by a general association between county admission levels and the ratios of population per physician, such as was found between these ratios and new case rates for cancer where the rank correlation, although not statistically significant, was +0.40.[4]

Because physician data were not available for county areas defined by population size, county ratios of population per physician were correlated with total county admission rates at ages 15 to 64. The resulting coefficient was low (+0.12) and not statistically significant. If the resources for medical consultation influenced the likelihood of the hospitalization of the mentally ill, the association did not appear to be simply a function of the number of doctors at hand.

No attempt was made at this time to measure possible regional variations in the public's recognition of mental illness. However, this factor is receiving attention in another analysis.

4. Population indices associated with county rates at ages 65 and over

Because of the findings at ages 15 to 64, the analysis at ages 65 and over paid heed to population size from the beginning. The correlation between the rates for areas 10,000 and over and those for areas under 10,000 was +0.60, higher than that at ages 15 to 64, and approaching statistical significance.

Based on areas with populations of 10,000 or more, county rates for ages 65 and over did not correlate significantly (+0.17) with those for ages 15 to 64, indicating that the indices found to be associated with the rates for the younger ages were not related to those for the older population.

The latter rates did, however, correlate highly and significantly (+0.79) with the average distance to the mental hospital serving the county. This association supported a widely held opinion concerning admission trends among the aged. It is often stated that rapidly increasing rates over the years, and urban excesses among the rates at a point in time, reflect a growing tendency, particularly evident in urban areas, to utilize mental hospitals for the custodial care of elderly dependents. Certainly, if the relationship between admission rates and the proximity of a mental hospital were a direct one, it must have been a function of those admissions for whom hospitalization was only one of several possible recourses.

Distance to a mental hospital was not correlated with county rates at ages 65 and over for county areas of under 10,000 population (+0.16). In fact, for these rates, as for the rates at ages 15 to 64, no indices were found which might aid in the interpretation of variations in the admission rates between the smaller urban and rural areas of the counties.

D. General Conclusions

This analysis was concerned primarily with the question of whether or not regional variations in first admission rates are simple functions of regional population difference in those variables known to influence the rate of admission to mental hospital. It appears that this is not the case and that regional differences arise on a much more complex basis. Although rural populations have lower admission rates than urban, county differences were not associated with the county variations in the proportion of urban dwellers. Although first admission rates among the single population exceed those among the married, county differences certainly did not arise on this basis. It appears, rather, that for urban areas, regional differences in admission rates at ages 15 to 64 are associated with a complex of variables descriptive of the kind of industrialization prevailing in urban centers. There is evidence that urban regional variations in rates at the older ages are in part a function of the availability of hospital facilities. No variables responsible for county variations among the smaller urban and the rural populations, at either age, were re-

[4] Calculated from data given in the 1951 Survey of Physicians in Canada and the 1953 Annual Statistical Report on Cancer in Ontario.

vealed by this analysis. This finding itself is provocative, but must be further investigated, for reasons already mentioned.

E. Summary

1. As one aspect of a project having as its objective the assessment of factors influencing the rate of admission to mental hospitals, an analysis was made of the distribution of these rates among the 14 counties of Western Ontario during the period 1950 to 1952. Its purpose was to determine to what extent geographic variations in the first admission level may be associated with those of other social characteristics.

2. County rates, ranked from high to low, showed a considerable range, and the county ranking correlated significantly with that for the period 1940 to 1942, pointing to the relative stability of the regional distribution of rates.

3. Attempting to find population indices with which the county rates were correlated, greatest progress was achieved when the admission levels at ages 15 to 64 and at 65 and over were calculated according to the population size, that is, for the segment of the counties with populations of 10,000 and over and for the smaller urban plus rural areas, separately.

4. At ages 15 to 64 county rates by population size did not correlate significantly with each other. Those for the larger urban areas did, however, correlate positively and significantly with the proportions of the general population who were married, under 65 years of age, and recent immigrants; and with the median wage of the head of wage earning families. These findings, it was felt, suggested a possible association between the admission level and industrialization.

5. At ages 65 and over the correlation between county rates by population size was again not statistically significant. Rates for the larger urban areas were closely associated with distance from mental hospital, pointing to the availability of hospital facilities as a possible influencing factor upon the admission level.

6. Among the smaller urban plus rural areas, no statistically significant correlations were found between the county rates for either age group and the available demographic indices.

7. None of the associations revealed by the analysis could be used as a basis for positive conclusions concerning causative relationships. However, variables were defined whose possible influence upon first admission rates can now be explored more intensively.

The writers acknowledge with gratitude the provision of special census tabulations for Western Ontario by the Population Section of the Demography Division of the Dominion Bureau of Statistics, the cooperation of the Mental Hygiene Division of the Ontario Department of Health, and the personal assistance given by Superintendents Dr. A. McCausland and Dr. B. H. McNeel, and by their staffs at the Ontario Hospitals in London and St. Thomas.

BIBLIOGRAPHY

1. Canada Year Book, Chapter 8. Ottawa: Dominion Bureau of Statistics, 1950.
2. Cohen, B. M., Tietze, C., and Greene, E.: Statistical contributions from the mental hygiene study of the Eastern Health District of Baltimore. *Human Biol., 11:* 485, 1939.
3. Dawson, W. R.: Relation between geographic distribution of insanity and that of certain social and other conditions in Ireland. *J. Ment. Sc., 57:* 571, 1911.
4. Dayton, N. A.: *New Facts on Mental Disorders.* Springfield, Ill.: C. C. Thomas, 1940.
5. Dorn, H. F.: The incidence and future expectancy of mental disease. *Pub. Health Rep., 53:* 1991, 1938.
6. Fandet, F. J., and Watson, R. I.: Relation between insanity and marital condition. *J. Abnorm. & Social Psychol., 30:* 366, 1935–36.
7. Faris, R. E. D., and Dunham, H. W.: *Mental Disorders in Urban Areas.* Chicago: Univ. Chicago Press, 1939.
8. Health Bulletin, Department of Health for Scotland. *Current Notes, 12:* 53, 1954.
9. Hyde, M. C., and Kingsley, L. V.: Relation of mental disorders to community socio-economic level. *New England J. Med., 231:* 543, 1944.
10. Malzberg, B.: Mental disease and the melting pot. *J. Nerv. & Ment. Dis., 72:* 379, 1930.
11. Mental Institutions, 19th Annual Report. Dominion Bureau of Statistics, Ottawa, 1953.
12. Redlich, F. C.: Social structure and psychiatric disorders. *Am. J. Psychiat., 109:* 729, 1953.
13. Tietze, C., Cooper, M., and Lemkau, P.: Schizophrenia and manic depressive psychosis and socio-economic status. *Am. J. Sociol., 47:* 167, 1941.
14. Wanklin, J. M., Buck, C., and Hobbs, G. E.: The trend of first admission rates to mental hospitals in Ontario, 1927–1946. *Canadian J. Pub. Health, 45:* 246, 1954.
15. Wanklin, J. M., Fleming, D. F., Buck, C. W., and Hobbs, G. E.: Factors influencing the rate of first admission to mental hospital. *J. Nerv. & Ment. Dis., 121:* 103, 1955.
16. Yule, G. U., and Kendall, M. G.: *An Introduction to the Theory of Statistics.* London: Charles Griffin & Co., 1950.

The University of Western Ontario
Faculty of Medicine
346 South St.
London, Canada.

A SYMPTOM ANALYSIS OF RURAL-URBAN DIFFERENCES IN FIRST ADMISSION RATES[1]

CAROL BUCK, M.D., PH.D., D.P.H., J. M. WANKLIN, B.A., AND G. E. HOBBS, M.D., M.P.H.

A. INTRODUCTION

In a previous report (1) we have described rural-urban differences in the rate of first admission to mental hospitals among the population of Western Ontario. The observed urban excess was consistent with the findings of other workers and, in spite of a number of special analyses of the data, was still open to two equally likely interpretations: 1) It represents a real difference in the incidence of mental illness, and 2) It reflects a difference in the demand for hospitalization. In the discussion of these results, it was suggested that a comparison of the symptoms of urban and rural patients might make it possible to move from this equivocal position. If a difference in demand for hospitalization causes the higher urban rates, the urban excess should be predominantly in the rate for cases whose symptoms are sufficiently tolerable socially that care outside of a hospital would be possible. The investigation of this hypothesis forms the subject of the present report.

B. METHOD OF ANALYSIS

A symptom analysis was applied to two groups of the Western Ontario patients who were first admitted to mental hospitals in 1950–52: 1) Patients aged 65 and over, subdivided further into three diagnostic groups: a) psychosis with cerebral arteriosclerosis; b) senile psychosis; c) other diagnoses. 2) Patients aged 45–54: a) males; b) females.

The first group was chosen because rural-urban differences were particularly marked among the aged. The analysis of the second age group was suggested by the results of the symptom analysis for the aged.

Rural and urban rates were calculated specific for symptom-type, so that for each population an admission rate for patients with Major Symptoms and an admission rate for patients with Mild Symptoms Only were obtained. Major Symptoms were defined as those which would make care outside of a hospital most difficult, and they are listed as follows: 1. Delusions (with or without hallucinations); 2. agitated and disturbed behavior; 3. threatening, destructive or assaultive behavior; 4. suicidal threats or attempts.

The rate for Mild Symptoms Only was obtained by counting cases with none of the above symptoms.

C. RESULTS

Figure 1 illustrates for age-group 65 and over the rural-urban differences in the rates for the two symptom-types. These are given separately for the three diagnostic groups. For psychosis with cerebral arteriosclerosis and senile psychosis the urban excess is predominantly in the admission rate for patients with Mild Symptoms Only, while the ratio of urban to rural rates for patients with Major Symptoms approaches unity (1.1).

The diagnostic group denoted as "Other" does not show the same trend, for here the urban excess is confined to the rate for Major Symptoms. Although small numbers precluded a detailed analysis of the individual diagnoses within this heterogeneous group, the proportion of urban admissions with Major Symptoms appeared to be greater for most of the diagnoses involved.

The latter observation led to the symptom analysis of age-group 45–54 where diagnoses comparable to those denoted as "Other" among the aged predominate. In this age-group, rural-urban comparisons of total rates (1) had shown a small urban excess among males and a small rural excess among females. When rates for the two symptom-types were calculated separately, the male urban excess was found to arise chiefly from the rate for Mild Symptoms Only, while the female rural excess remained only in

[1] From the Faculty of Medicine, The University of Western Ontario Medical School, London, Canada. This study was made possible by a Mental Health Grant from the Department of National Health and Welfare and the Ontario Department of Health.

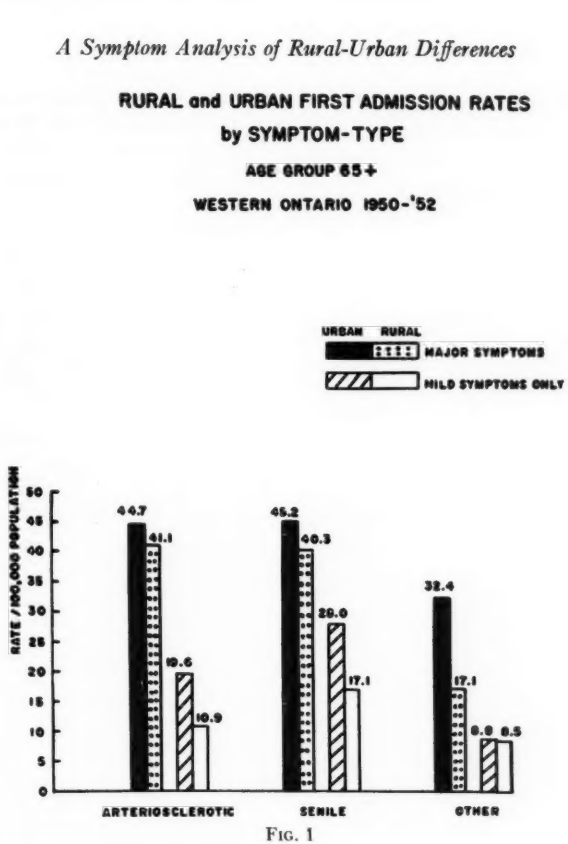


FIG. 1

the rate for Major Symptoms. Thus, the observations for this age group were consistent with those for the arteriosclerotic and senile psychoses among the aged.

However, before concluding that the urban excess, where it occurs, is the result only of the readier hospitalization of urban patients, another possibility had to be considered. If the symptoms which were considered "Major" arise late in an illness, the rates for Major Symptoms would really represent the rate of admission of advanced cases whose illnesses had progressed beyond the phase of mild symptoms. Were this true, an earlier hospitalization of urban cases could lead to the observations we have described, even if there were a genuine urban excess in the incidence of mental disease. For then, in addition to the effect of greater incidence, the urban rate for Mild Symptoms Only would be augmented further by earlier hospitalization which, in turn, could reduce the urban excess in the rate for cases reaching the advanced stage of Major Symptoms before admission.

Therefore, it was necessary to determine whether or not the symptom-types, Major and Mild only, differed appreciably in their average duration of illness. The results of this analysis are shown in table 1.

It is clear that within each group, the patients with Major Symptoms do not, on the average, have illnesses of longer duration before admission to hospital.

TABLE 1.—MEDIAN DURATION OF ILLNESS BEFORE ADMISSION (YEARS)

	Major Symptoms	Mild Symptoms Only
Ages 65 and over		
Arteriosclerotic	1.93	2.50
Senile	3.21	3.95
Other	1.04	0.96
Ages 45–54	1.90	1.83

D. Summary and Conclusions

Urban and rural first admission rates in Western Ontario, during the period 1950–52, have been broken down to give rates by severity of symptoms for the age-groups 45–54 and 65 and over.

The results of this analysis lead to the conclusion that for the age-groups studied, at least, the excess urban admission rate arises almost entirely from a greater urban tendency to hospitalize those cases whose symptoms could be tolerated in the community. An unexplained exception to this occurs in the rates for the mixed group of "Other" diagnoses among the aged.

The only alternative explanation for these observations would be that there is an essential difference in the symptomatology of rural and urban cases of mental disease, a possibility which seems highly unlikely.

It would appear, therefore, that the calculation of hospital admission rates in terms of symptomatology may be useful in distinguishing between real and apparent differences in the incidence of mental illness.

Bibliography

1. Wanklin, J. M., Fleming, D. F., Buck, C. W., and Hobbs, G. E.: Factors influencing the rate of first admission to mental hospital. *J. Nerv. & Ment. Dis., 121:* 103, 1955.

The University of Western Ontario
Faculty of Medicine
346 South St.
London, Canada.

POSTISCHEMIC DEMENTIA

A Case Report[1]

V. A. KRAL, M.D.

The effect of anoxia and hypoglycemia on brain functioning in humans as well as in experimental animals has been the subject of intensive studies in recent years. We are fairly well acquainted both clinically and anatomically with the immediate and short term effects of a sudden interruption of the oxygen supply to the brain (4, 11). Fisher's studies (1) on carotis-occlusion have clarified the clinical and anatomical picture of the slowly developing deprivation of the brain of a great part of its circulatory supply. Less understood are the long term effects, stretching over many years, of a sudden interruption of the brain circulation.

Gordon and others (3) describe a 21-year-old man who suffered circulatory arrest during a tonsillectomy. At the time of their publication, that is several months after the accident, the patient showed optic and tactile agnosia, defects of remote memory, disorientation, and concrete behavior. Freeman and coworkers (2) report on 3 cases of cardiac arrest all of which showed several months to 2 years after the accident severe intellectual and/or personality changes in addition to neurological symptomatology.

It is the purpose of this paper to report on a young man of previously average intelligence and behavior who at the age of 17 years suffered circulatory arrest during an abdominal operation. The history of this case during the first 12 months after the accident has been shortly described in a previous paper (6). The patient could be observed for another 5 years. During the later part of the 6-year observation period, secondary changes were noted in the patient's mental condition which, in spite of the relatively long time interval, still seem to be related to the circulatory arrest. It was felt that the case should be reported in order to stimulate long term reevaluation of similar cases by other workers.

[1] From the Verdun Protestant Hospital, Department of Psychiatry, McGill University.

G. M., a 23-year-old man, comes from a working class family. Both parents and two sisters are well-adjusted people. Neither of them has had a history of nervous or mental illness, nor was there any mental disease among other members of the paternal or maternal families.

The patient was a full term baby; the delivery and development were normal. He had whooping cough at 5 months of age, measles and scarlet fever during his second year, and mumps in his fourth year. There were no complications.

Patient started kindergarten at the age of 4½. He completed primary and two years of high school and passed every grade, slightly better than average. After he had left high school, he decided to pursue his aim to become an aircraft mechanic. He secured employment at the airport, and as he was not old enough to begin the work as a mechanic, he was given the job of handling baggage. He took every available opportunity to go up in one of the planes.

During childhood and adolescence the patient enjoyed playing with other children and being in a group. He was always interested in sports and played baseball, skated and bowled. He also enjoyed dancing and had friendly relationships with girls.

On the whole, G. M. has been a normal child and adolescent, enterprising, of average intelligence without any unusual problems, determined to achieve his aim to become an aircraft mechanic.

In February, 1949, in his 17th year, he developed a right inguinal hernia. On April 16, 1949, a herniorrhaphy was performed in spinal anesthesia. 0.14 mg. of pontocaine in 2.00 cc. glucose was used, and during the operation an injection of demerol equivalent to 1/16 mg. of morphine was given. During closure of the wound the heart stopped beating. A thoracotomy was performed, the heart massaged, and 1 mg. of procaine injected. After 4½ minutes, the heartbeat became normal again. An ECG taken at that time showed a normal tracing.

For the next two days the patient remained in coma in a state of decerebrate rigidity. Pyramidal signs were present bilaterally. The temperature was around 102°F., respiration irregular, pulse rate accelerated. After 48 hours the patient started to react to painful stimuli and to follow objects with his eyes. He was now semicomatose in a decorticate state, which was frequently interrupted by attacks of complete decerebration with unconsciousness. He was incontinent and had to be tube fed.

Six weeks after the circulatory arrest he was able to walk with assistance, to speak and to read a few words when stimulated. He was, however, somnolent in daytime, restless at night, still incontinent and unable to feed himself.

At the time of admission to the Verdun Protestant Hospital, 12 weeks after the accident, medical findings were normal. Neurologically, he showed three groups of signs: First, an extrapyramidal syndrome consisting of choreatic-athetotic movements of arms, hands and fingers, gross tremor of both hands and fingers, torsions of the neck and trunk, a cogwheel phenomenon on both upper and lower extremities and a masklike face.

Second, there was also a cerebellar syndrome; cerebellar gait, positive Romberg, hypermetria and cerebellar asynergy in getting up from the horizontal and sitting positions.

Third, a group of neurological findings was indicative of a lesion of the cervical segments of the spinal cord. They comprised bilateral atrophy of the shoulder and small hand muscles more on the right than on the left, with hypoactive deep reflexes on the upper extremities and bilateral Babinski signs. The remainder of the neurological examination was negative. The EEG showed a low voltage record throughout with some rapid activity.

The patient's mental condition was characterized by the following findings: In day time he lay quietly in bed without any sign of spontaneity but became restless during the second half of the night. He had to be fed and dressed and was incontinent of urine and feces. His sensorium was clear. He was, however, disoriented in all three fields, showed marked defects of remote and recent memory and severe impairment of immediate recall. There was amnesia for the operation and the following weeks including his transfer to this hospital in the middle of July, 1949. Although perception and recognition were unimpaired in all sensory fields, thinking was markedly disturbed. There were lack of motivation with poverty of ideas, inability to pursue a trend of thought due to distractability by external stimuli and poor concentration, perseveration, concreteness with impairment of abstract thinking. His verbal expressions consisted of short sentences given as answers to direct questioning. They concerned mostly his immediate needs. A tendency to simple confabulation was noticeable, mostly when the patient was asked to explain his stay in this hospital. He was able to read short sentences consisting of a few simple words, but he was unable to write or draw. Delusions and hallucinations could not be elicited.

Psychological examination showed an I.Q. of 45. In the Rorschach test the range of responses was narrowed; the form level, poor. He was unable to locate his references. There were considerable color naming, perseveration, verbigeration and some bizarre responses.

Systematic and sustained education and rehabilitation by the medical, nursing and Occupational Therapy staff were instituted. The patient also received 200 mg. of nicotinic acid three times a day for many months in an attempt to increase the intracranial blood supply (8).

Normal sleep rhythm was established in October, 1949, that is 6 months after the accident. At about the same time the extrapyramidal hyperkinesis had largely disappeared although some akinetic signs had become more apparent: the masklike face and the cogwheel rigidity, a lack of synkinesis of the arms in walking, a bent posture of the body. These signs disappeared by the end of December. The only remaining sign of the extrapyramidal syndrome, still present, is an athetotic posture of the fingers and hands. The spinal syndrome had continually improved; and by April, 1950, i.e., one year after the accident, the atrophy of the shoulder and hand muscles had completely disappeared, the reflexes were normal and pyramidal signs not elicitable. Improvement of the cerebellar syndrome had started later and remained incomplete. He still walks on a broad base with some cerebellar ataxia. There

also is still some imbalance in changing positions.

Improvement of the mental condition was less continuous and much slower than with the neurological signs. By November, 1949, 7 months after the accident, the patient had become clean in his habits, was able to eat by himself, and partly able to dress himself. A marked curiosity became noticeable. He would go to other patients to observe them, or walk into the examining office and look around there and take different objects into his hands. He would not speak spontaneously, however, and his answers to questions remained short and simple. Marked improvement was noted in reading, whereas writing and drawing improved only slightly. The I.Q. at that time was 50; the Rorschach test showed better control and less impulsivity. Stereotypy was diminished.

On psychiatric examination, immediate recall and recent memory were still impaired. He could, however, recall 3 or 4 out of 5 objects shown to him. He was disoriented in all three fields, would give his own age variously around 17–20, the date mostly as April, 1949. There was still complete amnesia for the operation and the following events. Confabulations regarding his admission to this hospital could still be elicited mostly to the extent that he had come here about one or two days or a week ago to have an operation performed. He denied ever having been in another hospital. A few experiences like home visits could be recalled, however; some names like the examiner's could be remembered. There was also loss of remote memories. He knew, however, that he used to work at the airport as a messenger and that he wanted to become a mechanic, but he could not give any details about his activities.

Thinking was disturbed by lack of ideation, distractability, perseveration and difficulties in conceptualization. He was, however, able to give some similarities and differences between concepts, like e.g., between river and lake, child and dwarf, and could understand and form some concepts like "parents", when speaking of father and mother, or "animals" when several pictures were shown to him. He could also perform some simple arithmetic and could name the king, the capital of Canada, of this province, etc.

For the following year the patient's condition remained unchanged. During this time he would take part in some occupational activities on the ward, but he constantly had to be reminded what to do. His actions were described as "clumsy" by the OT worker.

He would pick up an odd magazine and look at the pictures there, only to drop it after a short while. He continued with his curious explorations of his surroundings in his usual friendly but silent manner, never speaking spontaneously.

His emotional attitude was that of a flat euphoria without any signs of moodswings and with an apparently high frustration threshold. He would wait patiently until his meal was served and would not show any signs of anger or hostility when he happened to be the last patient to be served. He never got involved into any fights.

He was allowed regular weekend visits with his parents. On these occasions he would meet his sisters and their children, play with them and address them correctly by their names. He never objected to returning to the hospital.

Since the fall of 1950 a slow but steady decline in G. M.'s mental condition has become apparent. The first indication of this development was found in the Rorschach test. In November, 1950, this test revealed less mental control, more severe intellectual impairment, greater impulsivity and emotional deterioration. Clinically the lack of interest and initiative became more pronounced. He now would sit or stand around most of the time. His curiosity is gone, and he does not approach other patients or members of the staff as he used to, and he has lost all interest in OT work. He obeys orders as far as washing and dressing are concerned, is clean in his manners and is still able to eat by himself. In fact, the only interest he now displays, in the hospital as well as on his weekend home visits, is centered around his meals.

Memory function is more severely disturbed in all its aspects than in 1950. He is now unable to recollect more than 1 or 2 of 5 objects or to repeat more than 2 of 4 unrelated words. He knows the name of the examiner whom he has seen for 6 years, but he cannot name the nurses on the ward or any patient there. With the increasing impairment of immediate recall and

recent memory, his disorientation is becoming more marked. He finds his way around the ward on which he has been living for several years but is helpless when brought to another ward. He gives his own name correctly but his age as 14. He does not know where he was born nor the address of his last domicile. He gives the present date usually as the 4th of April, his birthday, and the year as 1932 (year of birth). He is unable to give the time of the day although he knows the time when a watch is shown to him. He knows that he is in a hospital but he does not know its name.

Impairment of remote memory also has become more severe over the past 3 years. Whereas 3 years ago he still was able to name the city and street where his parents are living, or to recall the names of his married sisters, he is unable to do this now. He does not recollect that he wanted to become a mechanic, that he had worked at the airport or why he left there. General information based on learned knowledge has become extremely poor. Whereas 2 years ago he could name the Queen, her predecessor, the capital of Canada, and the name of the province, he is unable to do this now. He does not know the number of months in a year, days in a week, cannot perform even simple additions and multiplications.

His distractability is increasing. On any auditory or visual stimulus he would immediately interrupt his verbal utterances, or any other activity, and would not resume them again. He needs to be stimulated again and again to continue with the description of a simple picture or in telling even the shortest event of his life. His associations are poor although not particularly slow. Recognition is prompt, and in the association experiment reaction words appear promptly after the patient understands what is expected from him. The lack of ideation and the poor quality of his associations become manifest, however, in the frequent reaction "I do not know" which he now gives to many test words as well as to many questions in an interview. Another disturbance of the associative process became more pronounced in the past two years: the tendency to perseveration. In the association experiment or in an interview he would repeat the test word or the first reaction word or any answer he has given to a previous question or even the words "I do not know" several times before another appropriate reaction can be elicited.

The level of conceptualization has now become concrete to the utmost. Concepts, even the familiar ones, are not formed. Although he recognizes his father and mother and knows their names, he is now unable to form the concept "parents". He does not form the concept "animal" when the pictures of four different species are shown or their species' names given to him. He is unable to give similarities or differences between concepts but always refers to them in relationship to himself. He knows that a lie or a theft is something "I should not do", a river a "water, I took a bath." It is interesting to note that only one year ago the patient was still able to give some difference correctly, e.g., between river and lake. He is unable to do so now.

The simple confabulations mainly pertaining to his stay in this hospital, which he produced during the first two years of observation, cannot be elicited any more. Instead, as the only spontaneous produce, serial speech can be observed occasionally like, e.g., naming the months of the year or days of the week or reciting the Lord's prayer.

Psychological testing revealed that his I.Q. had dropped from around 50 at the end of 1949 to 36 in the late fall of 1954. In the Rorschach the range of personality resources had deteriorated further with a corresponding increase in perseveration and minimal capacity for rational control and marked poverty of thought content. Signs of impulsivity had markedly decreased, and a loss of interest in the external world was indicated.

Whereas the E.E.G. record taken in July, 1949, was of low voltage with some rapid activity, the record in May, 1950, was of a much flatter type and showed some 4–7 per sec. slow wave activity. In 1951 the record showed 5–7 per sec. waves of low to moderate amplitude, particularly in frontal and central regions, with a tendency to come out in rhythmic bursts of about 5 per sec. Parietal and occipital activity was quite flat. This type of record remained unchanged until the beginning of 1955. The E.E.G. consultant, Dr. Hisey, concludes that the record suggests relatively little involvement of the cerebral cortex. He is of the opinion

that the abnormality is deriving from subcortical centers.

Discussion

Three major periods can be distinguished in the course of G. M.'s case. The first, from April 16, 1949, when the circulatory arrest occurred until the end of 1949, was characterized by continuous improvement in his neurological and mental condition. G. M. went through a state of deep coma and decerebration. This stage was followed by a semicomatose state of decortication, frequently interrupted by attacks of complete decerebration. This stage lasted for several weeks. At the end of this stage perception recognition and some voluntary activity began to appear. An extrapyramidal stage followed with some additional cerebellar and spinal symptomatology. During the approximately 5 months which this stage lasted, the neurological signs slowly receded until by the end of 1949 G. M. had reached a stage where his condition was characterized mainly by the psychiatric symptomatology.

The course during the first period is not unusual during the recovery phase after cerebral ischemia (5, 7, 10, 13). The relatively long persistence of the decorticate and the extrapyramidal stage seems to indicate the severity of the damage to the responsible cerebral structures, which, however, appeared to have been largely reversible. The great sensitivity of the Purkinje cells to ischemia could account for the cerebellar syndrome in our case. Signs of involvement of the cervical spinal segments have also been mentioned in the case of Howkins and others (5). They seem to be indicative of a particular sensitivity of the anterior horns of this region of the spinal cord to interruption of the circulation, at least in these two cases.

During the second period which lasted from the late fall of 1949 until the end of 1950 the patient maintained his improvement. It seemed to represent what at first appeared to be the permanent sequelae of the ischemic brain damage in our case: amnestic syndrome, disturbance of the thought process, emotional flatness, and a considerable loss of initiative. There was in addition a cerebellar gait, frozen athetosis of the hands and fingers, apraxia with impairment of writing and drawing. There were still some mental capacities left, however. Perception and recognition remained intact in all sensory fields, memory function was not completely lost, and there were still some initiative and interest left as expressed in G. M.'s curiosity and his willingness to take part in some ward and occupational activities.

Since the end of 1950, a third period of G. M.'s case started: A secondary decline in mental functioning. This decline became more apparent as the years passed on, particularly in an increasing loss of initiative and motivation, complete loss of abstract thinking with regression to utmost concreteness, increased distractability and deterioration of the remaining function of his memory.

The slow decline in mental functioning during this period, so long after the circulatory arrest and in spite of the continued interest on the part of the family and staff, is difficult to understand. Lidz (9) in his analysis of the amnestic syndrome points out how disruptive an influence the loss of recent memory has on the thinking and general behavior of a patient. There are no cues which he could use in his daily activities and in his thinking. If this loss lasts for some time, remote memories are lost, the patient becomes disinterested in his surroundings and increasingly more apathetic. It is conceivable that such a mechanism plays an important role in the decline of the mental capacities in G. M.'s case. Its effect must be felt even more strongly in a case with so much additional impairment of mental functioning.

Scholz (12), on the other hand, points out that there are to be found in experimental animals and in man exposed to cerebral hypoxia and oligemia focal lesions of vascular origin scattered throughout the brain. They are distributed in certain vascular areas as well as in systemic laminar distribution and consist in small areas of paling, homogenization or softening which are later replaced by glial sclerosis. Neubürger (11) studied the neuropathological findings in several cases of circulatory arrest with relatively short survival times. He observed parenchymal changes as well as vascular reactions. The latter appeared to depend on the length of survival. Whereas two hours after the circulatory arrest only stasis and escape of plasma was noted, endothelial swelling was present three days, and more pronounced proliferative vascular changes with consecutive

softening one week after the circulatory arrest. Although there is no report on record regarding the neuropathological findings in a case of circulatory arrest with a survival time comparable to that of our patient, there is a strong possibility that vascular lesions caused by the ischemia may be of importance in producing the progressive deterioration which we now observe in G. M.'s case. It is also possible that both factors, the psychological mechanism indicated by Lidz as well as circulatory changes, are jointly operative in this respect.

No attempt at localization of the functional deficits can be made in our case. It is interesting, however, that some functions usually correlated with parietal, occipital and temporal cortical areas were the first to reappear during the period of restitution and have so far remained unimpaired. There is no impairment of vision or hearing, no optic or auditory agnosia. Stereognosis is intact, the innner speech is not impaired, and there is no difficulty in orientation on the own body and no finger agnosia. This is in contrast to the findings of other workers who report on blindness (5), visual and tactile agnosia (2, 3), or difficulties in expressive and comprehensive speech functions in their cases (2).

Aside from an apraxia which seems at least in part responsible for his difficulties in writing and drawing, our case is characterized by the progressive decline of intellectual and facilitative functions. In the terminology of classical psychiatry the term "postischemic dementia" would seem appropriate to characterize both the progressive intellectual loss and its etiology.

Summary

A 6-year follow-up of a case of circulatory arrest which occurred in a boy of 17 during an abdominal operation in spinal anesthesia is presented. Three periods are distinguished in the course of this case: A restitutional period of about 7 months, a period of maintenance of improvement lasting for about one year, and a period of slow and steady decline lasting so far for 4 years. Some theoretical implications of these observations are discussed.

Thanks are extended to Dr. G. E. Reed, Medical Superintendent of the Verdun Protestant Hospital, for his valuable advice and permission to publish this paper.

BIBLIOGRAPHY

1. Fisher, M.: Occlusion of the internal carotid artery. *A. M. A. Arch. Neurol. & Psychiat., 65:* 346–377, 1951.
2. Freeman, R. V., Berger, L. M., Cohen, S., and Selle, W. A.: Major neuropsychiatric residuals following resuscitation from cardiac arrest. *J. A. M. A., 155:* 107–109, 1954.
3. Gordon, J. J., Sternlight, J. R., and Weaver, O. M.: Personality changes following cardiac arrest. *U. S. Arm. Forc. Med. J., 3:* 1469–1473, 1952.
4. Himwich, H. E.: *Brain Metabolism and Cerebral Disorders.* Baltimore: The Williams & Wilkins Company, 1951.
5. Howkins, J., McLaughlin, C. R., and Daniel, P.: Neuronal damage from temporary cardiac arrest. *Lancet, 1:* 488–492, 1946.
6. Kral, V. A.: Neuropsychiatric sequelae of cardiac arrest during spinal anaesthesia. *Can. M. A. J., 64:* 138–142, 1951.
7. Kral, V. A., and Gamper, E.: Körperdrehungen um die Längsachse in der Erholungsphase bei einem wiederbelebten Erhängten. *Monatsschft. f. Psychiat & Neurol., 84:* 309–323, 1933.
8. Lehmann, H.: Post-traumatic confusional state treated with massive doses of nicotinic acid. *Can. M. A. J., 51:* 558–560, 1944.
9. Lidz, T.: The amnestic syndrome. *Arch. Neurol. & Psychiat., 47:* 588–605, 1942.
10. Loeken, A. C., Haymaker, W., and Paulsen, D. L.: Stab wound of heart followed by temporary cessation of heartbeat with resuscitation by cardiac massage. Clinicopathologic study of one case of thirty days' survival. *Surgery, 26:* 745–755, 1949.
11. Neubürger, K. T.: Lesions of the human brain following circulatory arrest. *J. Neuropathol. & Exper. Neurol., 13:* 144–160, 1954.
12. Scholz, W.: Selective neuronal necrosis and its topistic patterns in hypoxemia and oligemia. *J. Neuropath. & Exper. Neurol., 12:* 249–261, 1953.
13. Strauss, H.: Strangulationsfolgen und Hirnstamm. *Ztschft. f.d. ges. Neurol. & Psychiat., 131:* 363–374, 1931.

Allan Memorial Inst.
1025 Pine Ave. West
Montreal 2, Quebec, Canada

SOME OBSERVATIONS IN POST-LOBOTOMY PATIENTS

H. C. KRAMER, M.D.

In a recent article (1), I described outbursts of involuntary, uncontrollable laughing spells of post-lobotomy patients which do not seem to induce gaiety in others. It looks at times as if something has been detached from an (up to now) integrated entity—the human personality. Since the article was written, additional operated patients have shown this reaction as well as some other unrestrained trends which I want to describe in this paper. Fitting them together we may one day be able to learn why this operation, which has shown good results in some cases, caused less beneficial side-effects in others.

It is well known that many psychotic patients use abusive and obscene language which they did not use when well. Yet, there is still a difference between the unrestricted kind of pouring out of abuse and obscenity observed in quite a number of post-lobotomy patients. Non-operated patients become abusive mainly when there is interference with their withdrawing habits or in response to delusions and hallucinations. It can be seen that they are suffering while using this type of expression and that it is a symptom of a pathological condition. As such, it seems to be recognized also by the other patients. The post-lobotomy patient propels these words out of his mouth continuously and aimlessly and without any apparent reason. He is usually aware of it, does not deny using it, while the non-operated psychotic patient is unaware of his doing it and frequently even denies it.

A patient operated on 5 years before, who has used this type of language most of the time stated, "Before the operation I was cranky and everything annoyed me, now things amuse me." The impulse to use this type of language becomes uninhibited in some patients just as the sexual urge does in others. A female patient discharged from hospital care a few years ago came voluntarily to the Mental Hygienic Clinic for advice. She was accompanied by her mother who stated that her daughter was steadily working on one job, adjusted well to outside and family life, had not caused any undue trouble except displaying a strong sexual drive which she had not shown previous to prefrontal lobotomy. On her way home from work she would approach any male person on the street, insisting that he have sexual intercourse with her. The patient was aware of her actions, knew about the dangers involved in such behavior, but was apparently unable to restrain herself. This patient was unmarried; yet a similar unrestrained sex drive could be observed in a married woman of middle age after prefrontal lobotomy. In the latter case the husband was able through understanding guidance to direct this uncontrolled urge into proper channels.

In using abusive and obscene language the post-lobotomy patient knows what he is doing, admits it, promises to control himself—only to burst out a few minutes later in the same kind of language. It is not directed against anyone in particular, and patients who have to share day halls and dormitories with this type of patient frequently complain about the incessant stream of abusiveness which puzzles them and which they usually are unable to ascribe to some particular person. It takes them aback, and it has not the vituperation and the pathological hostility of non-operated patients' abusiveness. It is often poured out, like projectile vomiting, which makes one think that it is cerebrally induced.

Of all the unrestrained tendencies seen in post-lobotomy patients, the use of abusive and obscene language is the last to disappear—if it disappears at all. Quite a number of patients do improve after the operation with regard to their behavior, their habits, their emotional stability, their mental functions and working ability, and their social contact. Yet in some of them the use of abusive and obscene language persists just as in some others the laughing spells persist. A patient operated on about 4 years ago has shown considerable improvement in all fields except in the use of abusive language. Having been

diagnosed, dementia praecox, paranoid type, one might think that she is still showing a symptom of her former condition. However, she did not display paranoid trends after the operation, mixed well with other patients (which she never did before), was neither suspicious nor mistrustful, aggressive nor assaultive. Yet the concept that the use of abusive language of post-lobotomy patients may still be linked to their former paranoid trends can be disputed. First of all, it is usually coupled with obscene language in post-lobotomy patients. Quite a number of them have not used this type of language previous to the operation. It also occurs in patients who have not displayed paranoid tendencies. For instance, a patient who has manifested typical hebephrenic features is using this abusive language together with obscenity one year after prefrontal lobotomy was performed on her. She has a good education and a refined background and probably had never used such language before—although she might have heard it from others. It might have been released from her subconscious mind after the operation. She has improved in many respects except for the use of this kind of language. She propels it forward with a smiling face and a kind of self-amusement. The other patients may become very angry at her and complain about it, yet they do not retaliate and seem even to tolerate it as time goes on just as they tolerate the laughing spells.

Some patients replace verbal abusiveness with abusive reactions, such as nose-thumbing, sticking out their tongues and so on, in the same manner as children. Here, too, the patient is usually aware of what he is doing and often resorts to this type of behavior when no one of authority is around. In fact it took us quite a while to verify the complaints of other patients and to catch the post-lobotomy patients doing these things. This rather childish display does not seem to be directed against anyone in particular and could be observed with post-lobotomy patients on different wards so that they were not influenced by each other. The climax of verbal and non-verbal abuse seems to be within the first and second year after the operation. It seems to subside in the following years, just as the beneficial results of the operation can be evaluated only after 2 or 3 years have passed.

Another inclination observed in a number of post-lobotomy patients is the tendency to steal. The terms "hoarding" or "taking away," as in non-operated patients, would not cover this kind of activity of the post-lobotomy patient. It really reminds one of the activity of the stealing magpie. Complaints have arisen from all wards where post-lobotomy patients are residing. Quite recently a patient in her early sixties, who was operated on with good results about 4 years ago, collected in no time 5 lipsticks, 4 compacts, bottles of perfume, jars of cream, and what-not after she had been raided on complaint half an hour before. Another time she slipped the collected items to a rather regressed patient who was found running around the ward with the objects in her hands. Unlike regressed psychotic patients, the post-lobotomy patients do not hoard trash or rubbish, and when they take things from garbage cans, which at times happens, they collect objects and not refuse. They mainly steal petty things which they cannot use and which are of little value to them. Thus the post-lobotomy patients apparently are able to distinguish between trash and objects in a kind of half-awareness. By stealing petty things, unselected and of little value to them, they resemble kleptomaniacs (who also grab petty objects of little value but never take trash or rubbish).

Post-lobotomy patients appear more easily suggestible than non-operated ones, and accept retraining and reeducation. Upon this kind of plasticity of their mental and emotional adjustment, the three R's of their treatment may be used, namely Retraining, Reeducation, and Rehabilitation. Retraining should start right after the operation, with regulation of their disorganized vegetative functions. Eating and eliminating habits can be retrained in a relatively short time. Coordination of movements, gait, and posture are the next to be retrained. It usually does not take longer than 6 to 8 weeks to retrain them. Reeducation follows by arousing interest in social contact, reading, writing, and entertainment. This is a longer proposition because of their lack of spontaneity. It can be overcome by a continuous and daily applied re-

education plan. Compulsive expressions and movements are usually well influenced by prefrontal lobotomy. One patient, operated on about 5 years ago, used to repeat the following words in quick and continuous succession, "You are a murderer and will be electrocuted in the electric chair." Very soon after prefrontal lobotomy was performed on her, we tried to make her say to people approaching her, "The Lord bless you and be with you." Some time after the operation she would mix up these two expressions until she learned to say only the latter one. She uses it with apparent joy and has forgotten the first one.

Rehabilitation is the aim to which all treatment should be directed. It needs much longer a period of time than retraining and reeducation and frequently has to be accomplished outside the hospital. However, the patients taking part in group therapy may be basically influenced toward rehabilitation. Their trends, wishes, and future plans can be discussed and their inclination toward rehabilitation stimulated. A female patient operated on about 4 years ago, in her late thirties, became a good domestic worker in the hospital but could not be released in spite of her improvement because she had a tendency to steal things from others and also to dig into the garbage can. She was the mother of two children and had a strong desire to take care of them. A rehabilitation plan was worked out which consisted in her taking part in group therapy where these tendencies were discussed in general. In addition she was reminded daily that stealing outside of the hospital would bring her into conflict with the law. Small presents which she apparently cherished were given to her. She has been released and discharged from the hospital for about two years.

We are not yet aware of the good and the evil forces which are released by prefrontal lobotomy. The only way to learn about them and utilize them in the progress of psychiatry is the exchange of observations and experiences. It was with this point in view that this article was written.

BIBLIOGRAPHY

1. Kramer, H. C.: Laughing spells in patients, after lobotomy. *J. Nerv. & Ment. Dis., 119:* 517–522, 1954.

PILGRIM STATE HOSPITAL
W. BRENTWOOD, L.I., N.Y.

A METHOD OF DISPARITIES FOR EVALUATING APHASIC DISTURBANCES

JOHN C. SNIDECOR, PH.D.

INTRODUCTION

The non-language behavior of the brain-injured has been discussed by many authorities since the time of Pierre Marie (1906). Such behavior ranges from the mild anxiety, rigidity, and perseveration observed in relatively stable aphasiacs to the "catastrophic" response described by Goldstein (2) which he considers a psychobiological phenomenon involving agitation, temper, aggressiveness, etc. In its extreme manifestations this defensive or avoidance behavior may involve a loss of consciousness. An excellent contemporary summary of such non-language deviations has been formulated by Wepman (3) in 34 categories, some of which necessarily overlap semantically and dynamically.

From the practical clinical view, anomalous non-language behavior, almost without exception, sets up barriers between residual cortical potential and objective demonstration of cortical function. Expressed in another way, the aphasiac, with due allowance for his state of physical trauma, appears to be in his way rather than on his way. Eisenson (1) discusses such barriers in a direct and usable manner under such heads as perseveration, rigidity and orderliness, concretism, withdrawal, etc.

METHOD

The clinical method herein described minimizes for some aphasic subjects the effects of non-language deviations described above. In presenting objects, pictures, verbal concepts, etc., one frequently meets complete or almost complete expressive blocks followed by withdrawal, negativistic behavior, irritation, crying, etc. To develop an easy-going, mildly competitive situation when such behavior is evident, a disparity of some degree is stated or inferred for each test item. The writer uses pictures and objects from a battery collected by himself with appropriate use of Eisenson's (1) items, the Stanford Revision of the Terman-Binet, etc. Examples of presentation of items are as follows:

"This is a game. I am going to show you some pictures, and I will say something wrong about each one. See if you can find the trick?"

1. Picture of wrist watch.
   Clinician: "What time does the alarm clock show?"
   Subject: Gives no response to time, hesitates, and giggles. "It don't wake me . . . bell . . . morning . . . alarm clock." No response to time.
2. Picture of ice-box.
   Clinician: "What would you like to cook in this nice white oven?" (Subject is a housewife.)
   Subject: "Children like cookies . . . cold . . . cold . . . Frigidaire . . . ice cube."
3. Picture of girl jumping rope.
   Clinician: "I like to see girls run, don't you?"
   Subject: "Jumps."

The next two items are from the Terman Revision vocabulary test. Obviously they cannot be graded as objectively as the test designers intended them to be. However, with this subject no usable response could be obtained by standard methods.

4. Clinician: "I like *oranges* in stew."
   Subject: "No, no, no, fruit not stew, for breakfast . . juice."
5. Clinician: "Do you *scorch* clothes when you iron for your family?"
   Subject: No verbal response, nods head, makes slight motion with hand as if ironing.
   Clinician: "Do you scorch the clothes with a teacup?"
   Subject: With some anger, "No, no, no, with . . . with . . . don't burn at all." (Laughs).

PSYCHOLOGIC RESULTS

The effectiveness of the technique can be inferred from the above descriptions. Its utility can best be demonstrated by two brief clinical reports stated below.

Mrs. A. is 47 years of age. She suffered a rather severe cerebro-vascular accident, right side involvement, and was subsequently unconscious for 3 weeks. After a period of 6 months she was referred by her neuro-surgeon to the writer. Blood pressure had dropped

materially, and initial spontaneous recovery was thought by the surgeon to be complete. Of German background, Mrs. A. had been a hard-working yet protected housewife, and she was now treated with even more protection and affection. The subject was emotionally labile to the point of crying several times when frustrated in the testing situation. No success was obtained with the verbal portion of the Terman. On a test of 50 simple pictures, 10 per cent of the identifications were accurate, or reasonably so. Four digits were repeated in reverse order, 6 in normal order; counting was unimpaired. Arithmetical problems, such as $7 \times 6 - 5$, could be solved quickly. Approximately one-half of all answers were in German and credited as correct. There was little delay in response. What appeared to be frustration and emotional clouding prevailed throughout the examination. When disparities were presented, the blockings were reduced and performance approximately doubled. Frustration appeared to be reduced. As a therapeutic device the technique is being continued with moderate success with assistance from members of the family.

Miss B. is a 64-year-old woman referred by the same neuro-surgeon following a 6-month period of spontaneous recovery. Her central nervous system impairment was similar to, yet perhaps more severe than that of Mrs. A. Miss B. had held administrative positions predicating a previous I.Q. of at least 120. In identification of pictures for name or function, only 10 per cent of the responses were satisfactory. Seventy-five per cent of the responses involved delays ranging from 2–3 to 30 seconds. Very simple materials could be read; response to pictures was not materially better in writing than in speaking. Arithmetic functions were markedly impaired. Miss B. was frustrated by delays and failures but evidenced little emotional blocking. When disparities were presented, the number of responses was more than doubled with increase in accuracy. Delay time was markedly decreased. The subject accepted the examination as a competitive game and laughed when she could react to subtle differences and occasionally said "that's stupid" when the differences were extreme. Arithmetic accuracy was only slightly improved by presenting false statements such as "7 and 5 is 13" or "6 times 8 is 46". This method of testing with Mrs. B. achieved a more effective evaluation than conventional methods. The device is being continued as part of a therapeutic program by a credentialled speech therapist working under the direction of the writer. Within the limits set by age and impairment the prognosis is fair to good. Quite incidental to this discussion, yet of interest, the subject is deriving pleasure from reading biographies written for children.

Conclusions and Discussion

For the subjects considered, and a limited experience with other cases, the technique of disparities has utility in evaluating aphasiacs and probably deserves research, perhaps as a basis for an M.A. thesis. For some subjects with approximately the same problems it is not productive. An informal yet systematic presentation of test items appears desirable. Although used primarily for evaluation, the technique is being utilized therapeutically with the same subjects with considerable success.

Summary

In order to work towards attenuation of emotional blocking, etc., in evaluating aphasiacs, test items are presented with a discrepancy between the object's name or function as represented in a picture or problem and verbal reference to it by the clinician. Two cases are presented to support potential utility in testing and in therapy. Systematic research should be a productive.

BIBLIOGRAPHY

1. Eisenson, T.: *Examining for Aphasics*, pp. 7–11. New York: The Psychological Corporation, 1954 Revision.
2. Goldstein, K.: *After-Effects of Brain Injuries in War*, pp. 70–74. New York: Grune & Stratton, Inc., 1942.
3. Wepman, T. M.: *Recovery from Aphasia*, p. 33. New York: The Ronald Press Co., 1951.

University of California
Santa Barbara College
Santa Barbara, Calif.

SOCIETY PROCEEDINGS

THE NEW YORK ACADEMY OF MEDICINE, SECTION ON NEUROLOGY AND PSYCHIATRY, AND THE NEW YORK NEUROLOGICAL SOCIETY

Joint Meeting, March 14, 1955

Morris B. Bender, M.D., Chairman, Section on Neurology and Psychiatry, Presiding

LOCAL TISSUE CHANGES IN VASCULAR HEADACHE OF THE MIGRAINE TYPE. DR. ADRIAN M. OSTFELD AND DR. HAROLD G. WOLFF.

Observation of painful scalp areas during migraine headache has shown that lowered deep pain threshold, local edema and alteration of tissue turgor were nearly constant findings. Hematomata and painful subcutaneous fluid collections were by no means rare. Furthermore, microscopic examination of the bulbar conjunctiva during headache revealed marked dilatation of all vascular structures with occasional venular hemorrhages. When the vasoconstrictor agents nor-epinephrine or ergotamine tartrate were administered, these evidences of local tissue alteration (i.e., edema and lowered deep pain thresholds) were eliminated more rapidly than occurred spontaneously. Edema of the bulbar conjunctiva has been terminated during nor-epinephrine infusion at a rate sufficient to attenuate and increase spasticity of the local arterioles. Dilated conjunctival vessels in headache have been shown to be more resistant to the constricting properties of topical nor-epinephrine than the vessels on the headache-free side. Local instillation of cortisone suspensions increased the sensitivity to nor-epinephrine. These observations confirmed the postulate that there was a substance or substances locally present in scalp areas involved with headache, whose release occurred during vasodilatation and whose withdrawal from the tissue was promoted by vasoconstriction.

Discussion.—DR. HAROLD G. WOLFF: Dr. Schottstedt and Dr. Ostfeld have made it evident that the troublesome gain in weight that so many patients with migraine exhibit before the onset of the headache and the state of hydration that some of us have tried to measure and analyze occurring during this period are not pertinent to the pain production itself. It is a concurrent manifestation found in many patients with migraine. Such persons gain in weight anywhere from two to as much as eighteen or nineteen pounds, the average being approximately five pounds, during the hours or days preceding the attack. It has been an attractive idea to some that this state of hydration has directly to do with the dilating of the vessels of the head so that they are caused to be painfully distended. These two workers have shown that it is extremely unlikely that this state of hydration has anything to do with pain, because patients accustomed to accumulate fluid before their attacks when given mercurial diuretics and diuretics of the Diomax type and who have thus been prevented from gaining in weight had just as many and severe headaches during their state of dehydration as in their hydrated state. Also it has been possible to show that increasing fluid loss with diuretics just before or at the beginning of an attack did not prevent the occurrence of the attack, even though the patient lost during the last stages of the attack all the fluid he had accumulated. Finally, in those who are in a phase of having many headaches the administration of pitressin in oil causing an accumulation of fluid and a gain in weight did not precipitate a headache. Hence, it is quite evident that there are two types of edema, one which is generalized and has nothing to do with the pain itself, but is a concurrent manifestation of the attack, comparable perhaps to extrasystoles, diarrhea, and constipation, and secondly, a local fluid accumulation at the site of the headache which occurs as the attack is beginning and progressing, and which is much like a local inflammatory reaction and relevant to the headache. Some of the properties of the local reaction you have just heard described by Dr. Ostfeld.

SUBDURAL HEMATOMA: ELECTROENCEPHALOGRAPHIC STUDIES. DR. JOSEPH G. CHUSID AND DR. C. G. DE GUTIÉRREZ-MAHONEY.

The electroencephalograms and clinical records of 55 of our adult patients with head injuries and verified subdural hematoma were reviewed; 34 of this group were studied preoperatively and 38 postoperatively. Classification of electroencephalograms on the basis of major abnormalities revealed that abnormal types and their incidence were similar in the preoperative and in the early postoperative (1 month) group. In 19 (66 per cent) of 29 unilateral subdural hematomas of the preoperative group, lateralization of major electroencephalographic abnormalities (slow focal or slow focal with diminished amplitude) coincided with the side of the hematoma. In the preoperative group of 34 patients, records were classed as slow diffuse in 11 (32 per cent), slow focal in 7 (21 per cent), slow focal with diminished amplitude in 14 (41 per cent) and normal in 2 (6 per cent). In the early postoperative group of 30 patients, records were classified as slow diffuse in 10 (33 per cent), slow focal in 9 (30 per cent), slow focal with diminished amplitude in 11 (37 per cent). In the late postoperative group (16 patients) tracings were made 3 months or later following operative evacuation

of subdural hematoma; in this group EEG was normal in 7 (44 per cent), slow diffuse in 2 (12 per cent), slow focal in 3 (19 per cent), focal spike in 3 (19 per cent) and slow focal with diminished amplitude in 1 (6 per cent).

Major studies dealing with preoperative electroencephalograms of adults with subdural hematoma reveal considerable disagreement in reported EEG findings and their significance. A variety of neuropathologic brain changes may occur in association with head trauma and subdural hematoma, which may significantly alter the electroencephalogram. In such patients cerebral swelling, increased intracranial pressure, brain hemorrhages or infarcts, laceration and contusion of brain, compression and distortion of brain and coup-contrecoup mechanisms may affect the EEG independently of the subdural hematoma.

In our experience cerebral angiography has been a more reliable and accurate test for the demonstration of supratentorial subdural hematoma in adults with head injuries than other commonly employed diagnostic tests (skull x-rays, electroencephalograms, pneumography).

Discussion.—Dr. Harry A. Kaplan: At Kings County Hospital we studied the EEG of 35 subdural hematomas, about half of which had postoperative as well as preoperative records. At least 80 per cent of these records were abnormal on the side of the EEG and at least 60 per cent had diminished amplitude of the waves, as well as a slowing of the activity on the side of the subdural hematoma. Postmortem examination of brains obtained from patients dying from a subdural hematoma revealed a flattening of the gyri of the brain opposite to the side of the subdural hematoma. The liquid hematoma seemingly maintained the rounded contours of the gyri directly underneath it. Microscopically, no abnormality can be seen in the cortex of either side. Drs. Browder and Rabiner, in a previous paper, have described swelling of the white matter of the brain beneath a subdural hematoma. We have been led to believe that this is the cause of the diminished and slow pattern of the EEG in cases of subdural hematoma. The EEG abnormality may persist from several weeks to a number of months after removal of the subdural collection.

Dr. Jack London: Have you any statistics regarding the number of negative EEGs in patients who show positive findings at operation?

Dr. Morton Nathanson: I would like to bring up one important aspect Dr. Chusid did not emphasize. For the most part, in dealing with subdural hematomas investigators concern themselves only with verified cases of subdural hematomas. Little is reported about the patients who show the same clinical picture as those with verified subdural hematomas, and are brought to the operating room, burr holes are made, and no subdural hematoma found. In evaluating the EEG, or any other clinical feature in relation to these patients, I think it behooves us to use as a control group those patients with negative explorations. Dr. P. S. Bergman, Dr. E. D. Friedman and I reported on such a series about two years ago. We found that the mortality and morbidity rate were almost identical in the two groups, and as for the EEG and other neurologic signs we found no significant difference between the two groups. Since Dr. Bergman is here I think he might mention something about the EEG changes.

Dr. Philip S. Bergman: We have compared the EEG in patients that had subdural hematomas and in those that were suspected of having subdural hematomas, but did not have them, and find that the subdural hematoma itself is not the explanation for the EEG changes. I noticed in Dr. Chusid's and Dr. Kaplan's remarks that everything seems to be pretty clearcut on the side of the lesion. Unfortunately at Bellevue I am not that good; I sometimes find the decreased amplitude is on the wrong side, and occasionally even the slow activity is more prominent on the wrong side. A third point I would like to make is that although there may be swelling of the subcortical white matter on the affected side, there could be a further explanation for the EEG changes. In a large number of cases we find changes in the brain stem, either small hemorrhages or edema, and the possibility that this is the source of some of the EEG changes has to be considered. This is perhaps important enough to be worth a study on animals.

Dr. Joseph G. Chusid: In our opinion, an awareness of the nature and extent of the brain pathologic changes that may be associated with subdural hematomas and with head injuries generally is necessary for understanding the EEG changes which may be associated.

The electroencephalogram was normal in 2 patients (6 per cent) of our preoperative group of 34 patients, both of whom had chronic unilateral subdural hematomas. In other reported series an incidence of normal records as high as 12 per cent has been noted.

Our presentation attempted to show the difficulties that one may meet if the EEG alone is relied upon for the diagnosis of subdural hematoma. The report of Levy et al (*J. Neurosurg. 9:* 588–98, 1952) was also mentioned, in which the control group consisted of head injury patients with negative explorations.

We particularly stressed that the EEGs of the early postoperative group resembled the preoperative group both in abnormal types encountered and in their relative incidence. This of itself should cast doubt upon the ability of the EEG to distinguish head-injured patients with subdural hematoma from those without.

The Use of Isoniazid in Multiple Sclerosis. Dr. John F. Kurtzke and Dr. Louis Berlin.

At the Bronx VA Hospital 30 patients with multiple sclerosis were given 300 mg. of isoniazid daily, and compared with 175 other patients identical in age of onset, age on admission, duration of present exacerbation, and length of hospitalization. The comparison group showed improvement in one-third, worsening in one-sixth, and no change in one-half. The isoniazid group showed 90 per cent improved and 3 per cent worse.*

Eight patients given isoniazid improved, relapsed when it was stopped, and again improved on a second

*The in-hospital course of these is published in *Am. Rev. Tuber. 70:* 577 (Oct.) 1954.

course of the drug. Four patients of a later group improved on doses of 500 to 750 mg. daily when 300 mg. produced no effect.

The 30 patients have been followed for 8 to 25 months since discharge. The 13 continued on isoniazid have maintained improvement. Five of the remaining 16 were given the drug again due to later exacerbations, and 4 of these have improved.

Twenty-one other patients were given isoniazid with improvement in 16 and no change in 5; 4 of these 5 had exacerbations of 3 to 8 years' duration.

Discussion.—Dr. Thomas C. Guthrie: The overall approach to multiple sclerosis therapy presented in tonight's report is highly commendable. By using a rating scale such as the one described, it should be possible to speed up the evaluation of drug effectiveness in this disorder. The rating scale provides a means of accurate and recordable observation which permits comparison of treated and non-treated cases. This method, however, must include alternate controls and a more detailed rating scale, one that would allow quantitation of motor and sensory function. I am sure the "double blind" method would also increase the accuracy of this study.

I would like to ask the authors how many times they have observed a patient relapse while on isoniazid.

In doses above 5 mgm. per kilo isoniazid is a neurotoxic agent. It has been reported to produce EEG abnormality, seizures in susceptible persons, and, more commonly, peripheral neuritis. Many of these adverse reactions can be prevented by the administration of pyridoxine and not nicotinic acid, as was originally thought likely.

Dr. Lawrence H. Gahagan: I would like to ask about the usual dosage; I noticed that large doses were given, but I should like to know what was the usual dose given.

Dr. John F. Kurtzke: I will answer the last question first. The usual dosage was 100 mg. three times a day. This seemed adequate in 90 per cent of our patients.

I am sure mathematically one could work out a probability of how many "exacerbations" and "remissions" must occur to be sure whether the intermittent use of the agent was doing something. We have not done this. But eight patients with two episodes; two of whom had more than two episodes, and all of these improved within a short time, is suggestive; that, plus the fact that the group not put on isoniazid after discharge showed five exacerbations as against this continued improvement.

We have seen no neurotoxic effects. We have given it to patients whose sensory distribution of dysfunction would correspond to a peripheral neuropathy, yet they improved. As to convulsive effects, we have observed none. EEGs were taken on admission and discharge on almost all of these patients, and even when the initial EEG was abnormal there was no worsening of the abnormality.

Parietal Lobe Syndrome Without Parietal Lobe Lesion. Dr. W. S. Battersby and Dr. H. P. Krieger.

The syndrome of hemispatial agnosia was found in 41 of 122 cases with cerebral disease and in none of 42 spinal cord control cases. This syndrome was demonstrated by routine and special tests. Particular attention was paid to visual field defects and to unilateral defects in spatial perception. Hemispatial agnosia was not found to be a unique expression of disease of the parietal lobe. The syndrome occurred with either left or right post-rolandic lesions provided there was enough dysfunction to produce a visual field defect and disorientation for time, place or person. For example, in one patient with a unilateral occipital pole sarcoma, hemispatial agnosia varied directly with the degree of disorientation, the field defect remaining constant throughout.

Discussion.—Dr. H. Houston Merritt: Dr. Battersby and Dr. Krieger have shown that visual inattention is not related solely to a specific lesion in one parietal lobe. I think most of us would agree with that conclusion, but I do not think we would be entirely convinced from the material presented that the symptom can occur in the absence of a lesion in the parietal lobe. Dr. Krieger did not present all of the cases, but I presume that most of them were brain tumors. It is difficult to draw any conclusion with regard to localization of function from the study of brain tumors because of the neighborhood effects and distant effects of these lesions. I would like to ask the authors if they had any lesions strictly localized to one lobe of the hemisphere to compare with these tumor cases. The authors emphasized the fact that this symptom may occur on either side of the body and that the preponderance of left-sided defects is due to the fact that patients with a disability on the right side of the body are usually also aphasic. We all routinely recognize visual spatial inattention when we approach a patient at the bedside and note that he does not pay attention to one speaking to him on the left side; we a think right away that the patient has a left hemianopia. We also take this fact into consideration whenever we visit the patient later: we never approach him from the hemianopic side, but always stand on his normal side because we know his attention is fixed on us on that side and that he is likely not to pay much attention to us from his affected side. The authors have not proved their point that the symptom of visual inattention can occur without a lesion in the parietal lobe. Regardless of that fact, they have clearly demonstrated that this phenomenon is not related to dysfunction of an isolated area, but it is accompanied by hemianopia in all cases, and disorientation and denial of the difficulty in a large percentage of the cases.

Dr. Morris B. Bender: Dr. Battersby and Dr. Krieger presented data on patients with brain tumors who showed sensory deficits and abnormal mental reaction, but there is some information which is as yet unpublished on the work done by Dr. Robert Jaffe and others in which some of the patients with brain tumor in the parietal lobe, or temporo-parietal region, did not show an organic syndrome and were not disoriented in space. Such patients when given sodium amytal intravenously may develop an organic psychosis and under this condition unilateral spatial deficit becomes apparent.

Dr. Howard P. Krieger: I do not think there is much to say after Dr. Bender's remarks, except to

stress the fact that, as in the last case shown, although the lesion was a permanent one in the temporo-occipital area, the syndrome came and went. I should like to stress the fact that we must have disorientation in addition to an organic lesion of a space-occupying type. We do not have any cases other than brain tumor cases with this syndrome, except for those cases which have generalized disease of the brain, and obviously they won't answer Dr. Merritt's question in regard to whether or not this syndrome occurs with what one might call a punctate type of lesion. We do not have any data on that.

THE PHILADELPHIA NEUROLOGICAL SOCIETY, THE NEW YORK ACADEMY OF MEDICINE, SECTION ON NEUROLOGY AND PSYCHIATRY

Joint Meeting, April 21, 1955

Axel K. Olsen, M.D., President, The Philadelphia Neurological Society, and E. Jefferson Browder, M.D., President, The New York Neurological Society, Presiding

Observations in Illusory Awareness of Bodily Parts.

Dr. Samuel Brock and Dr. Harold R. Merwarth, New York.

Three cases which illustrate remarkable abnormalities in body image perception are presented. (Because of limitation of space only one case is reported here. R. J. M.)

Case I is that of a very intelligent electrical engineer, holding a position of importance. He has had hypertension for many years, averaging about 180/110. On July 1, 1950 he developed a variety of oculomotor and other cranial nerve signs with ataxia, pointing to a brain stem lesion of thrombotic nature, involving a branch of the basilar artery. After some improvement there was a serious exacerbation in mid-September. Beside the considerable involvement of the cranial nerve innervations, there was a marked left hemiparesis and ataxia and a very severe left hemi-anesthesia with particular emphasis on the loss of proprioception. About the same time he developed the belief that the left arm did not belong to him, and at times belonged to his daughter. This finally gave way to the "feeling" that there was a parasitic left hand and a foreshortened forearm springing from the real forearm. These phenomena were associated with a great feeling of coldness in both the real and the spurious member. These body image disturbances fluctuated a good deal, became less evident as time went on, and disappeared altogether in the spring of 1951. All laboratory data were negative, including EEG. X-ray of the skull and spinal puncture were not done.

There seems little doubt about the gross anatomic diagnosis, namely, vascular lesions in the brain stem, particularly on the right side. There are a number of interesting points: (1) the patient's clear mentality and insight into the illusory nature of his parasitic hand, and the illusory posturing of the left upper and lower limbs at times; (2) the earlier attribution of the left limb to someone else, which disappeared, to be followed by the development of the parasitic part; (3) the cutting off of almost all sensation from the affected left limb and left side with the feeling of intense cold in the left "hand", i.e., the natural as well as the parasitic hands on the left; (4) the absence of any right or left confusion, aphasia or obvious apraxia; (5) the lack of any evidence of cerebral cortical disease; (6) the disappearance of the illusory awareness of the parasitic hand.

Discussion.—Dr. Gabriel A. Schwarz, Philadelphia: In looking over the three case histories, certain things seem to stand out as deserving of further discussion. In the first place, the laterality of the lesion is important. The second case was in the left cerebral hemisphere which I presume was the dominant or major hemisphere. The first and third cases suggested a lesion in the minor cerebral hemisphere. The literature to date has stressed the fact that disturbances in body image occur in the side opposite the minor hemisphere. Such a lesion is usually in the parietal lobe.

I would like to ask the authors the following questions: In their first case, while the lesion seemed to involve the brain stem, I wonder whether it is possible that a branch of the posterior cerebral artery might have been involved with subsequent damage to the thalamoparietal bundle and yet have given no evidence by electrocephalogram of its presence.

In the second case, do the authors have any explanation to offer for the phenomenon of incorporation occurring *bilaterally*, when the lesion was unilateral, in the left cerebral hemisphere?

In the third case, could the patient be made to recognize his left limbs as his own by feeling them with his right hand? Also in this third case, did he recognize both sides of the examiners, themselves, inasmuch as he had such a tremendous splitting of the extra-body hearing sphere?

Dr. Robert Jaffe, New York: In contrast to some of Dr. Brock's material, all the cases in our series who showed disturbance of body image or had difficulty in the appreciation of space had general disorientation. All of the patients showed some disturbance in visual function, and most showed some defects in somatic sensation, although perhaps only minimal disturbances in adaptation time, or disturbance in shape perception with bilateral stimulation.

Dr. Herald R. Merwarth, Brooklyn: There is little mention of the condition in the neurologic literature. I took occasion to investigate this question in

recent neurologic text books and examined ten of them. In three, anosognosia was not even mentioned. In others, the authors cited specific localizations. For instance, Nielsen places it in the connections between the thalamus and supramarginal gyrus. In the very recent three-volume Clinical Neurology edited by A. B. Baker, Foster states positively that the lesion occurs in area 40, Purves-Stewart places the defect in the thalamus. Russell Brain localized it to the supramarginal gyrus. It is discussed by Russell Brain, Wechsler, and the recent three-volume edition of Wilson. It is mentioned by deJong, and the 1911 edition of Oppenheim. As you can readily see, there is a wide fluctuation of opinion as to the nature of the condition and a possible anatomic site.

The question of anatomic involvement we know more about in the first case than in the others: this patient had a marked disturbance to cold; he could hardly tolerate it. We do see a disturbance of this type in spino-thalamic involvement in other parts of the nervous system, for instance, in the spinal cord, and in the brain stem, too. We believe there was no evidence to suggest a cortical involvement. There was no hypersensitiveness to pin-prick, as we see in thalamic lesion with hemiplegia dolorosa.

In regard to the question whether his proprioceptive sensation had returned prior to the return of motor function, I would like to stress the point which Dr. Brock made, and put it very clearly, viz, that this was almost essentially a sensory hemiplegia; there was motor involvement, but very little. All modalities of sensation were involved: pain, temperature. The proprioceptive modalities never did return, while pain appreciation did return in spots.

The other question regarding the phenomenon of incorporation of the extremities—that seems to be a psychological hazard. I am inclined to feel that it may be a carry-over of the so-called Gerstmann phenomenon (a projection, so to speak): the inability of the patient to recognize the examiner's fingers as not his own.

DR. BROCK: I want to emphasize what Dr. Schwarz has pointed out, i.e., that a sensory component may be an important factor in producing this curious syndrome in our Case I. In that case I would like to make it clear that this man's marked loss of proprioception persisted even though the parasitic hand finally disappeared. It might be argued our second case exhibited a Gerstmann's syndrome of some degree; the finger agnosia present on her first examination was almost gone at the second examination a short time later. She never showed any other change in her own body image. Just how the Gerstmann syndrome is to be correlated with her falsity of identification of the fingers of others placed in her palm, I do not know.

I would like to make the point that our first case was a very clear, intelligent, and cooperative individual who had no psychotic manifestations whatsoever, and I would like to emphasize that cases 1 and 2 had no denial of illness. In case 1 the man made up his mind that the parasitic hand was a neurotic manifestation, and so he would attempt to "brush it off", while still feeling all his incapacities, from a mild motor and a marked sensory defect on the left side; case 2 continued to be upset by her illness which she knew affected her brain.

THE EYE CENTERING SYSTEM. DR. MORRIS B. BENDER, New York. Paper to be published in *Arch. Neur. & Psychiat.*

Discussion.—DR. ERNEST A. SPIEGEL, Philadelphia: I was very much interested in following these experimental and clinical studies of Dr. Bender and greatly impressed by all the clinical observations and experimental data on which he bases his theory. The cardinal problem seems to me the relationship of the centering system to the deviational system, whether we deal here with two anatomically distinctly different systems, or with various functional states of one and the same system, depending on the state of excitation of the higher centers and the state of the peripheral neurones and on the parameters of stimulation. At the present time it seems to me hardly possible to decide these questions definitely, but even if further studies should show that a unitarian view should be accepted, it seems to me that Dr. Bender's hypothesis has led him to very important observations. I personally am particularly interested in the mechanism of vestibular nystagmus, and I should like to illustrate these problems by a few examples. If we elicit vestibular nystagmus with the eye originally in the central position, the labyrinthine component deviates the eyes upwards or downwards, and then a fast component in the opposite direction is superimposed. However, it never brings the eyes back to the central position; they remain in a deviated position. Another case is the rotational nystagmus; here we deal with a clockwise movement in one component and a counterclockwise one in the other component; or vice versa, but there is no centering in any component; any theory of nystagmus should of course explain all types of vestibular nystagmus.

A third example which is not so rare is if a patient looks in the direction of the fast component. For instance, you elicit a nystagmus by cold water irrigation of the left ear and let the patient look to the right side. In this way you produce a nystagmus to the right with a fast component to the right and the slow component to the left. We have here an example where the fast component is away from the center and the slow component is toward the center; in other words, we can hardly explain such a case by assuming that the fast component is identical with the centering system. It seems to me that Dr. Bender has devised a very ingenious theory, and I am sure he will continue to shed more light on many obscure neuro-ophthalmologic problems.

DR. NATHAN S. SCHLEZINGER, Philadelphia: In his presentation today Dr. Bender has again demonstrated his keen interest in many of the problems that have confronted the neurologist and ophthalmologist for a long time. The experimental observations that he and his co-workers have described since 1948 concerning mid-position or eye centering mechanisms have certainly been thought-provoking from a clinical standpoint.

When we consider the development of fixation reflexes it may be worth while considering whether the eye centering system is a purely subcortical involuntary

reflex mechanism or whether it is a cortical and at least partially voluntary mechanism.

Does Dr. Bender have any thoughts that would relate to the problem of concomitant strabismus and the various phorias and tropias?

With regard to optokinetic nystagmus, I find it difficult to follow the correlation between the quick component and the eye centering system since the experimental observation has been described as a slow rolling motion.

DR. MELVIN THORNER, Philadelphia: I should like to submit that there is, in man, an eye centering system which is quite independent of the existence of a nervous system. It is a mechanical system due to the elasticity of the capsule of Tenon and other orbital structures. If this be so, eye centering may then be accomplished either by this neurologically passive mechanism, or by the active and appropriate contraction of the extraocular muscles. To discriminate between the two, one would require action potential records either from the extraocular muscles or from the third, fourth and sixth cranial nerve complex. It may be that stimulation of a cerebral area, resulting in eye centering, is due merely to widespread inhibition of the nuclei of the third, fourth and sixth cranial nerves. In this case such stimulation merely sets the stage for a purely mechanical centering of the eyes to their position of rest.

DR. BENDER: I want to thank the discussors, and I must say I cannot answer many of their questions. I said that this was a theory. We are using this hypothesis to investigate the neuroanatomic basis for eye movements, and we have thought of the questions put by Drs. Spiegel, Schlezinger, and Thorner.

I want to make one comment about vestibular nystagmus. The fact that the nystagmus is increased in direction of gaze does not necessarily detract from the concept that the eyes tend to come back to their original position. If the term "center" is not accurate one may use "original position" or "midposition".

In reply to the question concerning optokinetic nystagmus, there is a quick component which returns toward the center and a slow deviational which follows the stripes. The eye centering mechanism corresponds to the quick component.

As for Dr. Thorner's question, we are well aware that the eye centering might be a complete relaxation of eye movement. It is suspected that in many conditions the centering movement is an inhibitory effect but there are instances in which the centering is an excitatory act. We think that eye centering is the result of a combination of both excitation and inhibition.

TOTAL REMOVAL OF ACOUSTIC NERVE TUMORS. DR. J. LAWRENCE POOL AND DR. ARTHUR A. PAVA, New York.

Total removal of an acoustic nerve tumor is the procedure of choice if it can be safely done, for according to the literature it removes a 50 per cent chance of recurrence within 3 to 5 years which in turn carries a 25 to 60 per cent risk of fatality after a second or third operation. Total removal (author's series) also restores nearly 80 per cent of the patients so treated to a useful capacity in life, which is a better percentage than that for subtotal removals.

Pre-operative contra-indications to total removal include cerebral arteriosclerosis, vascular hypertension, myocardial damage, gastric or duodenal ulcer, or debilitation. Operative contra-indications to total removal in one stage are extreme central or midline extension of a large tumor, and excessive vascularity or adherence of the tumor at the site of its contiguity to the brain stem. Careful study of these cases also suggests that a significant though temporary failure of vital signs before or during the operative removal of tumor tissue is often a bad prognostic sign that should warn the surgeon against total removal. Total removal of necessity implies the threat of facial nerve palsy, owing to the fact that the tumor nodules within the internal auditory meatus must be removed, which generally makes it difficult to save this nerve. However, facial-hypoglossal nerve anastomoses almost always restore effective voluntary control to the paralyzed side of the face.

Altogether then, each case must be carefully considered not only before, but again at the time of operation, so that everything possible be done to preserve the lives and functions of patients harboring these most difficult of all brain tumors. In a word, while total removal is clearly the procedure of choice, it should not be attempted in the presence of any of the contra-indications just described.

Discussion.—DR. FRANCIS GRANT, Philadelphia: The removal of an angle tumor is the most difficult operation in neurosurgery. If you can take an angle tumor out, you can feel you are a good neurosurgeon. The problem I think is just about as stated by Dr. Pool. Some of these tumors we can extirpate, and some we cannot get out. The tendency most of the time I have found is to try to do a little bit too much.

I am frank to admit I have never saved the facial nerve. Olivecrona says he can, and Dr. Pool says he can, so I should be able to, but I must say I never have. Postoperativeiy hypoglossal-facial anastomosis will give a satisfactory result. My feeling is that the sacrifice of the facial nerve is rather a cheap price to pay for the complete removal of an angle tumor.

DR. RUDOLPH JAEGER, Philadelphia: I have adhered to the surgical philosophy of total removal of cerebellopontile tumors and I do not at present recall any instance of subtotal or intracapsular removal of these tumors. In several instances I must have left tumor tissue where it had eroded deeply into the temporal bone through the internal acoustic meatus. However, I cannot remember a second operation in such a case.

The facial nerve has, in my recollection, seldom been saved. I recall recently total removal of bilateral tumors of considerable size with complete preservation of this nerve on one side. Spino-facial anastomosis has always been performed for correction of the facial paralysis. A motion picture shows the movement of the face following spino-facial anastomosis.

PROFESSOR NORMAN DOTT, Edinburgh, Scotland: I would like first to bring to you, Mr. Presidents, and members of the two Societies, greetings from the neurologists of Europe. As you know, we are watching

over there, with the greatest interest and admiration, advances that are made here, and we gain great inspiration from them. Many of you know my personal debts over here to Harvey Cushing many years ago; and I should like to thank you very much, as a vagrant neurologist for the moment, for your hospitality in allowing me to attend this meeting. I have listened with great interest to the papers. In respect of the last paper by Dr. Pool that I am invited to discuss, I would say that in Britain we agree entirely with his general attitude to these tumors. It occurred to me that I might mention three relevant matters. Dr. Pool mentioned the difficulty of carrying out the ideal operation of complete removal in patients who showed before operation some instability. I think that to some extent we have been able to meet that situation by draining the ventricles externally for several days before such an operation, by frontally inserted catheters.

The second point that I might mention is that I observed with the greatest admiration the spino-facial and hypoglossal-facial anastomoses results. We have followed your example there. I have had occasion five times to repair the facial nerve somewhat more directly in removing an acoustic tumor. At times the facial nerve can be quite clearly seen, and in the interest of complete removal it may be deliberately cut across and the tumor completely removed. In this circumstance one can apply nerve grafting. The graft is sutured to the stump of the severed facial nerve close to the brain stem, brought out through the cerebellar access, pushed forward under the sternomastoid muscle, and some 90 days later, when the growing fibers are nearing the lower end of the graft, then the second suture is made at the back of the parotid gland anastomosing the graft to the distal part of the facial nerve.

DR. AXEL K. OLSEN: The points you mentioned, Professor Dott, may be small, but at least, so far as I am concerned, they are very important.

DR. POOL: I do not want to let this opportunity go by without thanking Professor Grant for his kind and stimulating remarks, and also Dr. Jaeger.

Dr. Jaeger asked about the 12th nerve versus spinal accessory nerve anastomosis. Dr. Stookey some years ago in his book on Peripheral Nerve Surgery went into the theory extensively as to why hypoglossal nerve anastomosis is more successful on theoretical and practical grounds because the hypoglossal is linked with the cortical mechanism for speech through voluntary face and mouth motion much more closely than the cortical representation of the spinal accessory nerve.

In reply to Professor Dott, I have drained the ventricles preoperatively in one case with great success as to ease of total removal of the tumor followed by a complete recovery. We should undoubtedly do this more often.

CONCERNING HYDROCEPHALUS ASSOCIATED WITH LIPOIDOSIS OF THE CENTRAL NERVOUS SYSTEM. DR. STANLEY M. ARONSON AND DR. ABRAHAM RABINER, Brooklyn, N. Y.

Eight cases of infantile amaurotic family idiocy were institutionalized for the major part of their clinical course. These cases displayed all the classical features of Tay-Sachs' disease, but in most instances survived for periods of time beyond that considered representative of the illness. This clinical prolongation was considered a reflection of active nursing care rather than any inherent variation in the disease. Weekly head circumference determinations were recorded in all cases. After approximately 18 months of overt illness, rapid cranial enlargement emerged in almost all patients. Multiple pneumoencephalograms disclosed a moderate internal hydrocephalus. Review of autopsy material from this actively studied group, and of an additional 9 autopsied cases, showed some correlation between illness-longevity and brain weight. Beyond approximately 18 months of recognized illness, brain weights tended to increase above the normal for that particular age. The degree of actual megalencephaly increased in the more protracted cases.

Pathologic studies of the protracted cases showed a massive degeneration and demyelination of the cerebral white matter, grossly similar to cases of Schilder's disease. The neurocellular damage incident to the dystrophic process resulted in axonal and myelin loss in the underlying cerebral white matter. The increase in brain weight was felt to be a reflection of secondary and exuberant medullary gliosis and massive associated edema. The internal hydrocephalus was attributed to a diffuse meningeal fibrosis incident to the continuous discharge of abnormal lipid components into the subarachnoid space.

Discussion.—DR. ALEXANDER SILVERSTEIN, Philadelphia: I should like to express my appreciation for the opportunity of listening to the instructive, stimulating paper by Drs. Aronson and Rabiner. The authors present convincing evidence in a large series of cases of amaurotic family idiocy that there is a correlation of illness-longevity to the megalencephaly. This results from a massive degenerative change in subcortical white matter secondary to the neurocellular damage. I have had no personal experience with such cases of megalencephaly in Tay-Sachs' disease, nor have I discovered a similar series of cases in the literature.

I should like to ask whether histo-pathologic studies were made of various viscera, such as pleura, osseous system, liver and spleen. Chemical analyses of the lipids in various organs would also have been enlightening. In view of the mention I heard of chemical studies, were chemical analyses made of the various organs other than the brain?

DR. CHARLES RUPP, JR., Philadelphia: Dr. Aronson and Dr. Rabiner have reported their findings in respect to hydrocephalus and megalencephaly in Tay-Sachs' disease. As they point out, their findings are not exactly new, but they have correlated certain facts that have been scattered in the literature for some time. Lichtenstein states that subsequent to the loss of nerve cells there is a secondary nerve fiber degeneration which possibly accounts for the diffuse sclerosis found by the authors.

There is possibly another clue as to why these cases resemble Schilder's disease. In the authors' cases there has been a longer survival period than usual, and there has been more time for lipid accumulation to occur.

Consequently demyelination and glial repair with edema have progrsseder futher than usual.

Dr. Aronson: As Dr. Rupp stated, the megalencephalic aspects of Tay-Sachs' disease are not unique to this presentation.

Insofar as our cases are concerned, the question was asked whether or not they are actually instances of Tay-Sachs' disease, or perhaps represent some aberrant form related to the juvenile or adult forms of amaurotic family idiocy. I wish to re-emphasize that all of these cases showed all the classical features which we feel are basic to Tay-Sachs' disease. In all cases there was macular degeneration, a finding not generally present in the juvenile form of amaurotic family idiocy. These children would perhaps have died at the anticipated time were it not for the excellent nursing care they received, which may explain why they lived longer than usual.

Insofar as the other organs are concerned, with the exception of the two cases of Niemann-Pick's disease (which died 14 and 15 months respectively after the onset of their illness), the organs were essentially normal by all histologic and histochemical procedures.

Demyelination and secondary necrobiosis were most prominent in the cerebral white matter corresponding to cortical areas where diffuse plenary destruction of the neurones was evident. The parieto-occipital and frontal regions showed the most devastating changes.

THE SUPERSENSITIVITY OF CHRONICALLY PARTIALLY ISOLATED CEREBRAL CORTEX AS A MECHANISM IN FOCAL EPILEPSY AND IN LOBOTOMY. DR. FRANCIS ASBURY ECHLIN, New York (Read by title).

In one series of monkeys blocks of cerebral cortex were neuronally isolated or partially isolated and in another frontal lobotomies were performed. Seven to 10 months later 0.2 per cent acetylcholine applied to the cortex caused wave and spike discharges selectively from the isolated or partially isolated areas. 0.2 to 0.75 per cent acetylcholine produced similar discharges selectively from the frontal lobe of the lobotomized animals. The isolated and partially isolated areas were also supersensitive to electrical stimulation and to intravenous metrazol.

The findings so far are in keeping with Canon's Law of Denervation which provides a plausible explanation of the mechanism underlying focal cortical epilepsy.

Lobotomy causes a profound change in the physiologic chemistry of partially isolated cells (a supersensitivity) in the frontal lobe and probably elsewhere. It is therefore suggested that the clinical effects of tobotomy are probably, in part at least, the result of hlis supersensitivity.

NEW YORK ACADEMY OF MEDICINE, SECTION ON NEUROLOGY AND PSYCHIATRY, AND THE NEW YORK NEUROLOGICAL SOCIETY

Joint Meeting, May 10, 1955

Morris B. Bender, M.D., Chairman, Section on Neurology and Psychiatry, Presiding

INTRACRANIAL TUBERCULOMAS—A REVIEW OF CLINICAL FEATURES AND ANALYSIS OF PRESENT-DAY TREATMENT. DR. WILLIAM A. SIBLEY AND DR. JOSEPH L. O'BRIEN, Neurological Institute.

Intracranial tuberculomas behaving as brain tumors, rare in the United States at present, still represent a large proportion of intracranial mass lesions in many parts of the world. A review of 18 cases seen at the Columbia-Presbyterian Medical Center since 1933 revealed that the clinical manifestations of these lesions are not unlike those of other brain tumors; diagnosis is difficult prior to operation or autopsy. Sixteen of the cases were adults, in sharp contrast to most other series, and seizures and evidence of increased intracranial pressure were the outstanding clinical features in the majority of the cases. Prior to the advent of antituberculous drugs the mortality rate from attempts at extirpation of these tumors was prohibitively high due to the development of tuberculous meningitis; decompressive operations were therefore considered to be the indicated form of therapy. The over-all mortality rate in 102 collected cases treated with a combination of surgical excision and post-operative streptomycin therapy was about 22 per cent, representing a marked improvement in results since the introduction of specific drug therapy. The development of tuberculous meningitis in the postoperative period has been prevented in almost all cases by streptomycin therapy continued for 60 to 90 days after operation. Treatment for longer periods of time, in the absence of meningitis, probably has no definite advantages and would enhance chances of drug toxicity and development of bacteriologic resistance. Isoniazid and para-aminosalicylic acid should be used in addition to streptomycin, although few cases so treated have been reported as yet. Frequent spinal fluid analyses should be performed for several months postoperatively. If clinical symptoms or spinal fluid evidence of tuberculous meningitis supervene, then, of course, treatment is continued for much longer periods in accordance with present concepts of the therapy of tuberculous meningitis.

THE SYNDROME OF LESION IN THE CORPUS OF LUYS: CASE PRESENTATION. DR. HUMBERTO CRAVIOTO, Bellevue Hospital (N. Y. U. Division).

A case of typical right acute hemiballismus, in a 69 year old hypertensive man, of 60 hours duration, with postmortem verification, is presented. The abnormal, violent involuntary movements began suddenly on February 1, 1954 and stopped 60 hours later when the patient went into deep coma. The abnormal movements lessened in intensity with deepening of the conscious level and also with intravenous administration of amobarbital sodium (2.5 per cent solution). After 150 mg. of this drug the movements showed a decided change and by 10 minutes after (250 mg.) they stopped completely, to return 20 minutes later with equal characteristics as prior to the injection. The patient died 5 days after the onset of hemiballismus. At autopsy a discrete recent hemorrhage (1 x 0.5 cm.) on the left corpus of Luys was found, a halo of blood infiltration impinged on the internal capsule and in Forel's field. The literature on the subject is summarized and discussed. Emphasis is made of the fact that in cases of hemiballismus, lesions of the corpus luysii are not always present, but that destruction of the afferent and/or efferent connections of such nucleus also gives rise to the syndrome. Microphotographs of the gross hemorrhagic lesion are presented.

CLINICAL ASPECTS OF CEREBRAL ANGIOGRAPHY IN CHILDREN. DR. CHARLES M. POSER, Neurological Institute.

During the last 10 years, 90 children were subjected to cerebral angiography. 131 cerebral angiograms were performed, of which 117 were by the percutaneous route, including all the children in the series under the age of 2 years. All but 2 were performed under general anesthesia and most of the procedures done during the last 2 years were done by the neurologists. Cerebral angiography was of definite diagnostic value in 43.5 per cent of the cases.

No fatalities were encountered. Morbidity and complications, including temperature elevation and fall, increase in pulse rate, blood pressure fall, pupillary inequality, petechiae, convulsions, neck hematomas, respiratory difficulties and hoarseness, were present in 35 per cent of the procedures, but were all mild and transient. Complications believed to be due specifically to angiography were present in 20 per cent of the patients. Comparison with an equivalent series of lumbar pneumoencephalograms in children revealed a morbidity of 49 per cent. It is felt that the morbidity and complications are affected in some way by general anesthesia and premedication. No definite relationship between the amount of contrast material used, the age of the patient and the incidence of complications could be established.

Cerebral angiography is a useful and relatively harmless procedure in children when the proper precautions are taken and the indications are definite. The percutaneous technique is suitable for children of all ages.

Discussion.—DR. DANIEL SCIARRA: I would like to ask a question within which may be hidden a comment. So often we find in doing arteriograms that we are doing them after the pneumoencephalograms have already showed pathology, and we hope the arteriogram may contribute in addition, or we do the arteriogram first, and then we have to do an air study to get the information we want. Here Dr. Poser has shown that in 44 per cent of the patients pathology is indicated on the arteriogram. I would like to know in what percentage of patients does the arteriogram add anything that has not been elicited by other diagnostic procedures.

DR. DAVID R. CODDON: I enjoyed your paper Dr. Poser. Recently I have completed a study of the complications occurring in carotid cerebral arteriography among some 191 patients on the Neurology and Neurosurgery Services of The Mount Sinai Hospital. Our subject group was comprised of adults. We regarded complications as any objective detrimental clinical change occurring in the patient within the first 24 hours following the procedure. Elevation of temperature, and variations in pulse rate were so common that they were not regarded as complications. Complications occurred in 14 per cent among the 220 attempted procedures. It is important to emphasize that in only two patients were the complications of a permanent nature, i.e., hemiplegia. There were no fatalities in this study. Most of the procedures were done by the percutaneous technique. It is important to mention that in our study analysis of the data failed to reveal any factor that could be used to predict the occurrence of a complication in any one patient.

DR. POSER: To answer Dr. Sciarra's question first, in about two-thirds of our cases the patients had both an air study and an angiogram. Our time tonight was quite limited, so I was not able to present the material we have on correlation. However, from the slide shown it is obvious that (1) in the patients who were suspected of having an angiomatous malformation and the patients presenting with subarachnoid hemorrhage, the angiogram was the procedure of choice, and (2) a large number of patients had cerebral angiography because they presented with convulsive disorders, and it was thought that a vascular malformation might be an irritant, and those patients had cerebral angiography, which was followed by pneumoencephalography, which of course adds nothing to the diagnosis. I cannot answer Dr. Sciarra's question completely. I think the important thing we have learned from the study is that if cerebral angiography is done in many cases you can make the diagnosis of cerebral atrophy and in that case pneumoencephalography or ventriculography is not necessary. One of the reasons for bringing up this material is to try to help the pediatricians. Cerebral angiography is probably just as easy to do as a pneumoencephalogram.

PNEUMOENCEPHALOGRAPHY WITH MINIMAL WITHDRAWAL OF CEREBROSPINAL FLUID. DR. PAUL SLOSBERG AND DR. MURRAY BORNSTEIN, The Mount Sinai Hospital.

This paper is the second report on a new technique of performing pneumoencephalography, viz., the injection of gas but the withdrawal of as little cerebrospinal fluid as possible, with resultant reduction in the severity of the patient's post-pneumoencephalogram

reaction. The original report concerned 35 cases but no controls had been used at that time.

The present study included 84 additional cases, of which 64 were "minimal withdrawal" cases and 20 were controls, i.e., cases in which more than 5 c.c. of cerebrospinal fluid was removed). Reductions in the test group in (1) duration of post-pneumoencephalogram headache and (2) post-pneumoencephalogram temperature elevation are shown graphically in comparison with the reactions in the group of controls. Vomiting and chills also occurred less frequently in the test group than in the control group. In the control group two deaths occurred. However, one case had undergone surgery before death. The only mortality in the test group occurred in a patient whose clinical course had been progressively downhill *before* pneumoencephalography and who *after* pneumoencephalography underwent both arteriography and surgery (a glioblastoma was found) before death (which occurred on the fourth post-operative day).

Quality of the pneumoencephalographic x-rays was unchanged. Diagnoses confirmed by pneumoencephalography included supratentorial neoplasms as well as a neoplasm of the posterior fossa (verified by surgery). Three patients in the test group and one patient in the control group had cerebrospinal fluid pressures of 200 mm. or more. One patient in the test group had papilledema with hemorrhages and another patient in the test group had "questionable papilledema." No control patient had papilledema.

This study supports the contention of the previous paper, viz., that reaction to pneumoencephalography is reduced by this new technique.

Discussion.—Dr. Sidney Carter: I did not hear a description of the x-ray results. Did you get good pictures when only very small amounts of fluid were removed? Is this not an unusually high incidence of mortality, 1.7 per cent after PEG,—three deaths?

Dr. William Amols: I notice that the authors use caffeine for the treatment of post-PEG headache. I would like to ask if they have found this drug helpful and what, if any, they consider to be the rationale for caffeine in this particular condition?

Dr. Humberto Cravioto: We have been doing the same procedure routinely in Bellevue on the Third Neurological Division. We have done a large number of pneumoencephalograms; out of a number of more than 100 I do not remember more than one death about 27 hours after the pneumoencephalogram in a man who *at operation* had a large glioblastoma in one hemisphere, which extended to the other; in that case, as in most cases, we got excellent pictures. In some cases we have done the procedure two or three times, and have had no complications. It has been amazing to see the patients after a pneumoencephalogram done with this method walk out of the x-ray room and go to the wards in excellent condition; many of them eat two to three hours later and have no headache at all. Usually the headache comes after the first 10 c.c. have been injected, is frontal and at times severe, but in most cases it goes away in ten minutes or so, or diminishes greatly. Most cases, too, only require 35 to 50 c.c. of air with no fluid removed.

Dr. Lawrence Levy: I am interested to know what is the spinal fluid pressure in the patient who had 100 c.c. of air introduced without having any fluid removed. I feel fluid should be removed and air introduced in about equal amounts. It seems to me that 100 c.c. is a lot of air to put in and not take any fluid out.

Dr. Slosberg: In answer to the first question concerning the quality of the pictures, the Radiological Department sees no significant difference in the quality of the pictures, whether fluid is taken out or not.

As for the deaths, these all occurred in patients who had pathology; two had glioblastomas, and one had a vascular malformation.

Dr. E. Jefferson Browder: Some 20 years ago we were investigating the relationship of cerebrospinal fluid pressure, blood pressure, pulse rate and respiratory rate under a variety of conditions. In an attempt to sustain an increase in cerebrospinal fluid pressure various substances were injected into the lumbar thecal sac. Among these was air and in one instance approximately 700 c.c. were introduced within an hour. No cerebrospinal fluid was withdrawn. At the end of the period of an hour the cerebrospinal fluid pressure was the same as recorded before the injection. This subject had a normal ventricular system by pneumoencephalography. I wonder how air as well as cerebrospinal fluid escapes so rapidly from the subarachnoid spaces? It would seem that most of it enters the blood stream in some manner.

The Use of I_{131} in the Localization of Brain Tumors. Dr. Fremont C. Peck, Jr., Dr. Howard S. Dunbar and Dr. Bronson S. Ray, New York Hospital.

I_{131} in the form of radioactive iodinated human serum albumin was administered to 220 patients with suspected intracranial disease. Ninety of these patients were proved to have brain tumors. Of this group 54, or 60 per cent, were correctly localized with the scintillation counter; 40 per cent were missed. There were 77 cases of tumors of the cerebral hemispheres in the series; of these, 53, or 69 per cent, were correctly localized. Pituitary and posterior fossa neoplasms could not be accurately localized by this technique. It was also found that 7 of 9 chronic subdural hematomas gave significantly high counts over the side of the lesion. In the total series of 220 there were 8 false positives, a percentage of 3.6 errors.

Increased vascularity of the tumor tissue and changes in the blood-brain barrier in the region of the lesion are thought to be responsible for the increased uptake of the tracer substance in the cases studied. The radioactive isotope technique outlined is considered a useful adjunct in the diagnosis of cerebral mass lesions, and gives the best results with meningiomas, glioblastomas, metastatic tumors and subdural hematomas.

Discussion.—Dr. Philip S. Bergman: In the 40 per cent of cases classified as "missed", what does that mean? Does it mean that the method said there was no tumor, or the method said it was in the wrong place, or what? That makes a big difference, it seems to me.

DR. MORRIS B. BENDER: Did you use this method on patients with intracerebral hematomas, and did this method help to localize these hematomas?

DR. PECK: The cases that were described as "missed" in this group were for the most part normal readings. There were a few in which the localization was off. I recall one case of a deep right frontal parasagittal meningioma which gave a higher count just to the left of the midline, and that was considered missed, but almost all of them were normal records.

We have not in our series any cases of intracerebral hematomas which were tested by this technique.

NEUROLOGICAL COMPLICATIONS OF THE NON-CHROMAFFIN PARAGANGLIOMAS. DR. LABE SCHEINBERG, Neurological Institute.

The non-chromaffin paraganglia include the carotid body, the glomus jugulare and paraganglion tympanicum, and the glomus intravagale. These structures are associated with the parasympathetics of cranial nerves and are chemoreceptor in function. Tumors arising from these structures are histologically similar and behave alike clinically. They tend to be highly vascular and friable, are often multicentric, and give rise to neurological symptoms and signs by pressure necrosis and local invasion.

Occurring most often in the glomus jugulare and paraganglion tympanicum of the middle ear, they result in tinnitus, deafness, otorrhea, vertigo, spontaneous hemorrhage, and later gradual involvement of the lower cranial nerves. Other neurologic signs are rarely seen. The cases from the literature are reviewed as to the incidence of neurologic complications, and one typical case seen at the Neurological Institute of New York is presented in detail.

The rare tumor of the glomus intravagale usually gives rise to involvement of the lower cranial nerves only, while carotid body tumors almost never result in neurologic deficit.

Most of the tumors are seen in middle-aged females, while males are affected, and it occurs in all age groups. These tumors tend toward chronicity with gradual progression, though the prognosis for life is generally good.

The treatment of these tumors prior to extensive involvement of the nervous system is excision, but after there is invasion only irradiation is indicated.

It is of importance to the neurologist to differentiate this tumor from more common neurologic entities giving rise to almost identical clinical features, since surgery after neurologic involvement is commonly hazardous.

PROGNOSIS OF THE SUICIDAL PATIENT. DR. LEONARD M. MOSS, New York Hospital (Westchester Division).

To determine those factors influencing the prognosis of the seriously suicidal patient, an analysis of the case histories of 50 such patients treated at the New York Hospital-Westchester Division was undertaken. In all cases there was no doubt of the intensity of the suicidal drive.

A characteristic reactivation of suicidal tendencies was found in 90 per cent of all patients. This occurred when the patient was markedly improved and came into contact once more with the environment in which the illness began. The suicidal drive had, therefore, not completely subsided even when the patient was relatively free from tension and was considered convalescent.

Suicidal gestures, including successful attempts, took place in the majority of reactivations. Management of this phase is the most crucial aspect of therapy and the chief prognostic factor for the present illness.

Recovery required a major change in the life situation. Only 3 recovered cases returned to the same environment in which the illness arose without fundamental changes in either marital relationships, employment, or psychosexual orientation. The patient remained a potential suicidal risk until this alteration in the life situation developed as a probability.

Follow-up period averaged 6½ years for recovered cases, 4 years for all cases. Fifty per cent were recovered and 20 per cent much improved at the time of last follow-up. Eleven patients died by suicide. Of these, 7 were removed from the hospital against advice during the acute phase or while in a period of reactivation in the convalescent phase. These deaths occurred without 2 to 4 months after removal from the hospital and represent casualties of the reactivation. Four patients remained permanently hospitalized.

Of the 37 considered recovered or much improved at the time of discharge, 3 had a recurrence of suicidal tendencies. Six of the 37 who were considered successfully treated had subsequent psychiatric disorders. None proved to be suicidal risks. Prognosis was found to depend on the psychotherapy and compensatory changes in the life situation, rather than on any particular factor in the patient's background or a descriptive aspect of his illness. Successful therapy does not prevent subsequent illness, but can eliminate the prospect of the patient's once again acting out self-destructive fantasies.

Discussion.—DR. GEORGE H. HYSLOP: In what psychiatric classifications were these patients placed? What proportion represented constitutional psychoses?

DR. MOSS: Of the 50 patients included, 35 per cent were in the manic depressive category; 25 per cent were psychoneurotic; 22 per cent were schizophrenic; 10 per cent were involutional psychoses, and the remainder either psychoses with psychopathic personalities, or psychopathic personalities without psychoses. Approximately 60 per cent were considered psychotic on admission.

THE EFFECTS OF SERPASIL ON HOMEOSTASIS IN PATIENTS PRESENTING PSYCHOSOMATIC SYMPTOMS. DR. STANLEY BERNSTEIN AND DR. THOMAS DETRE, The Mount Sinai Hospital.

Preliminary clinical observations on 21 patients treated with serpasil at the psychiatric service of The Mount Sinai Hospital were presented. The diagnoses from a psychosomatic point of view included ulcerative colitis, hypertension, asthma and neurodermatitis. All patients received a uniform dosage of 5 mg. serpasil I.M. for a period of 3 weeks, supplemented by

5 mg. serpasil P.O. After 3 weeks the I.M. dose was gradually replaced by oral medication which amounted to approximately 5 times the I.M. dosage. In this study the I.M. serpasil seemed to be much more satisfactory than its oral equivalent.

From a psychological point of view, the administration of the drug has been associated in all cases with the production of nightmarish dreams and a gradually increasing tolerance toward anxiety. This led to what the authors call a "peeling off process," that is, a reintegrative mechanism in which the most recent symptoms and defenses mechanism are replaced by previously existing ones.

It was felt that through a greater production of dreams, patients release sufficient amounts of anxiety to permit their entering into a meaningful therapeutic relationship.

Discussion.—Dr. Martin A. Green: I think that the evaluation of drugs in general is very difficult, especially so with these types of patients. Whether the patient is actually reacting to a specific drug, or to the attention being paid by the physician to certain symptoms is always a problem. I think before any conclusion can be drawn a control series or a substitution technique on the same group of patients should be done, and the person evaluating the results should not be aware of what the patient is receiving.

Dr. Bernstein: I think you are absolutely right.

A Schizophrenic Child Becomes Adolescent. Dr. Leon Tec, Bellevue Hospital.

Some juvenile delinquents show no schizophrenic symptoms, but are, nevertheless, basically schizophrenic. This observation comes from the follow-up of schizophrenic children who lost their schizophrenic symptoms when they approached puberty. Their behavior and psychopathology at adolescence was that of a psychopathic personality or of a character disorder.

As an illustration a case is cited of a 15 year old boy who was under psychiatric observation from the age of 8. At 8 he was at Bellevue Hospital on the Children's Psychiatric Service and was found to be schizophrenic, both by the psychiatric staff and the psychologist. He had hallucinations, introjected bodies and poor reality testing. Confabulations and contaminations were noted on the Rorschach.

At the age of 15 he was seen again, this time for robbery, repeated truancy and delinquent behavior. The patient had none of the schizophrenic symptomatology and the only outstanding feature was his complete lack of guilt. He did not remember any of his productions of childhood and could not revive them even under sodium amytal or under hypnosis.

Thus, it is our impression that childhood schizophrenia plays an important role in the etiology of some cases of juvenile delinquency.

The disappearance of all the schizophrenic symptomatology at puberty makes the correct diagnosis of these cases impossible unless we are in the possession of their preveious records (if such records exist).

Thanks to the cases that were observed in childhood and followed, we are able to get better insight into the mechanism and etiology of delinquency.

Discussion.—Dr. Lauretta Bender: I only want to emphasize the importance of the observation which Dr. Tec has reported here. It is only within the current year that in Bellevue we have fully realized the significance of this change of the schizophrenic child through puberty into adolescence. We have seen a clearcut diagnostic picture of schizophrenia in childhood and in puberty the child seems recovering when he is under therapy, or the diagnosis is changed, as it often is at Rockland State Hospital or other institutions. Then in mid-adolescence they become impulsively and dangerously aggressive, and at the same time feel they are not responsible for their acts, and they do not show evidence of guilt or anxiety, and even seem to enjoy what they are doing. Our more extensive follow-up indicates in early adulthood there is a recurrence of a frank psychosis, thus a large number of our childhood schizophrenics turn up in State hospitals as clearcut schizophrenics or dementia praecox in their twenties, whereas in early puberty they were considered to be promising a complete recovery of childhood symptomatology, and in mid-adolescence they were found to be psychopathic in their behavior. This represents a very important view for many of our present problems and throws considerable light on the life course of some early schizophrenics and later anti-social behavior.

Delusional Reduplication of Parts of the Body Following Insulin Coma Therapy. Dr. David N. Graubert, Hillside Hospital, Glen Oaks, N. Y.

A 34-year old man was admitted to Hillside Hospital with a 6-week history of withdrawal, visual and auditory hallucinations, neglect of personal appearance, and aggressiveness. He was placed on insulin coma therapy and showed no change until his 19th treatment. On this occasion he was noted to have a right hemiplegia, hemisensory syndrome, hemianopia, and "spatial inattention." He denied having anything the matter with his arm but said there was an additional right arm in his bed which did not work. He referred to it as an "extra arm," "a third arm," and "a bootleg arm" or personified it as a "stranger", "this intruder" and "that fellow." At the same time he was euphoric, loquacious, friendly and showed disorientation for time. His neurologic symptoms and the delusional reduplication disappeared within several hours. For a few days afterward he was depressed, complained of feeling "depleted". This gradually subsided and in the following weeks the patient became quiet and cooperative, made attempts to socialize, and pressed for his discharge. He no longer showed any of the psychotic manifestations which brought him into the hospital. He was discharged from the hospital and is doing well on the outside while continuing on psychotherapy.

The discussion centered around three main points of interest: (1) the neurologic picture as a complication of insulin coma, (2) the symbolic importance of reduplication of body parts with special reference to the work of Weinstein and Kahn, and (3) the implication of the subsequent changes in the patient's mental status for an understanding of the mechanism of insulin coma therapy.

Discussion.—Dr. Richard M. Brickner: Nothing

was said about the underlying neurologic diagnosis. I wonder whether that might not be important in the genesis of the entire clinical state. I think more emphasis should be placed upon it.

DR. LAURETTA BENDER: In our experience in electric shock treatment with children we have observed a tendency to duplicate parts of the body, but rather along the longitudinal axis; that is, there will be several heads or several bodies, and this occurs during the actual course of the shock treatment, immediately after an actual convulsion. Later on the whole body image concept of the child is reorganized in a more primitive fashion than before the shock treatment started, permitting the child to mature along the more natural course. This was published in a paper by Dr. W. R. Keeler and myself in the American Journal of Orthopsychiatry, in 1952. This case reported tonight seems to come much nearer to the phenomena of anosognosia which Paul Schilder reported in his body image studies. I would hardly look upon it myself as a dislocation of parts, but rather as a way of accounting for a defective limb in a patient who has hemiplegia which he denies and which has been reported under the heading of anosognosia.

DR. GRAUBERT: Reduplication of body parts is actually a more widespread phenomenon than one might think. Therefore it is not surprising that it is found in other conditions of altered brain function, such as those referred to by Dr. Bender. The basic point that we are trying to make about this phenomenon is that while it has traditionally been described as a disturbance of body image or, in the work of Critchley, it can be understood as a symbolic expression representing some motivation or need. It is certainly related to anosognosia and is usually used to facilitate expression of denial of illness.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Meeting of April 21, 1955

Dr. Erich Lindemann, President, presiding

CYSTICERCOSIS CEREBRI: A DIAGNOSTIC AND THERAPEUTIC PROBLEM OF INCREASING IMPORTANCE. JAMES C. WHITE, M.D., WILLIAM H. SWEET, M.D. AND EDWARD P. RICHARDSON, M.D.

The recent literature on infestation of the human brain by the larval form of T. solium indicates that this condition is becoming a neurosurgical problem of increasing importance. This is true not only in countries with poor hygiene but also here and in England.

Cysticercus cellulosae in the brain gives rise to varied clinical manifestations which may resemble brain tumour, pseudotumour (cerebral hydrops), Alzheimer's or Pick's disease, and luetic meningitis.

Three case histories are reported of patients with this condition seen in the Massachusetts General Hospital during the past two years. One had lived in this country for 36 years.

The first two represent the favourable type of solitary cysts which resemble a brain tumour by production of localizing symptoms and signs. In each of these obstructive hydrocephalus was erroneously attributed to a midline tumour in the posterior fossa. Relief was obtained by removal of the obstructing cysts from the fourth ventricle. In the second, complicating recurrent adhesions blocking the outflow of cerebrospinal fluid necessitated further surgery to re-establish ventricular drainage.

The third patient suffered from epileptic seizures due to multiple calcified cysts scattered through both hemispheres. No operation has been necessary, as the fits have been controlled by anticonvulsant medication.

Two other varieties of intracranial cysticercosis have been described. The first, with multiple intracerebral cysts, often leads to fatal oedema of the brain; the second, to serious hydrocephalus from the presence of racemose cysts blocking the basilar and ambient cisterns.

In order to diagnose and treat this disease effectively it is necessary to know its common clinical forms, to realize its frequency in certain countries where hygiene is poor, and to bear in mind that the parasite may cause trouble many years after its entry into the blood stream.

Valuable diagnostic tests are eosinophilia of the blood and spinal fluid, a positive complement fixation to an alcoholic extract of porcine Cysticerci by the cerebrospinal fluid, and roentgenographic techniques.

Discussion.—DR. DAVID GAMSU: Cysticercosis is quite prevalent in South Africa, particularly in the Bantu population. A number of cases were admitted to hospital because of epilepsy on whom the diagnosis was obvious. These cases revealed scattered calcified areas easily visible in the brain, characteristic of cysticercosis. Frequently areas of calcification were too numerous to count. These patients were put on anticonvulsant medication and were left alone unless something gross developed. Surgery is unjustifiable where only one focus is removed and hundreds of others are left. Follow-up is impossible in the Bantu population, for once treatment is over, the patient returns to his tribe and is not seen again.

In regard to the question of multiceps multiceps, I have seen four cases in the white population which presented collections of cysts in the form of grape-like clusters in the posterior fossa. The first three were operated upon and did fairly well at the time of operation, but later symptoms recurred. At autopsy multiple cysts were found in the subarachnoid space around the tentorium. The fourth case, a man of 40, presented a posterior fossa lesion, with increasing headache, papilledema, vomiting, vague cerebellar signs, increased pressure, and increased protein. Investigation showed him to have hydrocephalus, with a posterior fossa obstructive lesion. At operation, grape-like cysts were found in the cisterna magna and fourth ventricle and were removed. A torkildsen procedure was performed. The patient recovered and left after two weeks, apparently quite well. He returned in three months with recurrence of symptoms, but without the cerebellar signs. Pressure 500. The site of incison had started to yield. I opened the incision again, but found no evidence of recurrence of the cysts. He had one big arachnoid cyst, with free connection between the fourth ventricle and the lateral ventricles, viz. a communicating hydrocephalus. The pathological lesion was therefore at the tentorial notch interfering with the return of the cerebrospinal fluid. It was felt that an attempt to do an anterior third ventriculotomy would result in failure, as no drainage could be expected. I did an arachnoido-periteneostomy with a tube leading from the lumbar subarachnoid into the peritoneal space. He did well postoperatively, the pressure remaining at almost zero. When last seen the patient was perfectly well. Apparently there is no systemic treatment to get rid of the cysts, or to stop them from recurring.

DR. SOLOMON HAKIM: The paper of Drs. J. C. White, W. H. Sweet, and E. P. Richardson, Jr. gives us a very good idea of the problem of cysticercosis of the brain. I would like to summarize some of the conclusions that Dr. Alejandro Jimenez and I arrived at in several cases we saw in Bogota, Colombia. A reprint of our first four cases was published in 1954.[1] We have since examined and studied nine more cases. The importance of this disease in Boston, of course, is not due to its high incidence in this vicinity, but rather to its occurrence in patients who come to this medical center

[1] Cisticercosis del Encefalo en Colombia. Revista del Hospital de San Juan de Dios, Bogota, Volumen II, Numero 6, 1954.

for neurological and neurosurgical care. For this reason, it is perhaps worthwhile to be familiar with this troublesome disease.

Cysticercosis of the central nervous system should be regarded as a chronic, multi-focal condition, especially when it is located in the parenchyma. The classifications that have already been proposed by Griesinger, Sato, Hanneberg et al, Tyczka and Ran-Prussakowa, and finally by Stephen and Chorobski are based mainly on the clinical symptoms and the location of the parasitic cysts, resulting in many groups, and thus become controversial and not practical.

A more practical and simple classification is both desirable and possible. We preferred to divide our cases into three groups:

1. The intraparenchymatous group, in which the cysts were mainly located supratentorially;
2. Cysticercosis of the cerebrospinal fluid pathway, which was primarily intratentorial, and
3. The mixed forms.

The reason for this grouping is that the parasitic implant can be either in the parenchyma, in the cerebrospinal fluid system, or in both.

With respect to our cases, in the first group, the cysts were localized both in the cortex and in the white matter, and in many sites throughout the brain. These cysts gave rise to symptoms, such as convulsions, mental impairment, and hypothalamic disturbances, depending upon the function of the involved areas. In all of our parenchymatous cases, there were multiple cysts and in three of this group, serial skull x-rays at six-month intervals revealed the presence of additional calcified cysts. Another case revealed no calcification in the initial x-rays, but because the patient had convulsions and by biopsy was shown to have proven skeletal muscle cysticerci, was kept under close observation and maintained on anticonvulsant therapy. An X-ray taken one year later revealed calcifications in the right occipital lobe.

In the second group, the cysts were localized anywhere in the cerebrospinal fluid system, either subarachnoid or within the ventricles. The circulation of the cerebrospinal fluid was interfered with, resulting in a hydrocephalus.

An important feature worth emphasizing is that in the intraparenchymatous group the cysts tend to develop into a spherical form as they grow, perhaps due to concentric pressure from the surrounding tissue. In the second group, in which the cyst lies within a free space, it grows in a form molded by the surrounding structures. If there are many cysts in the subarachnoid space, for example at the base of the brain, they resemble a flattened and distorted cluster of grapes, and are called "racemose". This is a purely descriptive word, and adds nothing about the nature of the condition. The racemose form is entirely due to the effect of the confining space within which the cysts have to grow.

In the third group, the mixed form, the posterior fossa symptoms usually dominate the picture.

The diagnosis of cysticercosis is not always easy. Eosinophilia is often present, but it is usually transient, there are many other conditions which produce it, and when the parasite dies and becomes calcified, eosinophilia is no longer present. The cerebrospinal fluid is not consistent. It varies from a normal fluid to one which contains an increase in total protein, with or without mononuclear cells, depending upon the severity of invasion and localization of the parasites. The electroencephalogram, in most cases, is diffusely abnormal, despite clinical symptoms which point to a focal lesion and x-rays which demonstrate a single calcified cyst, meaning that perhaps there are multiple involved areas.

When the cysticerci are localized in the posterior fossa, we found, as Asenjo and Arana pointed out, that ventriculography reveals a symmetrical dilatation of the lateral ventricles and third ventricle, with partial filling of the fourth ventricle, and presence of air in the cisterna magna. This picture is the result of partial occlusion of the fourth ventricle and aqueduct, allowing air to pass into the cisterna magna. This ventriculographic picture is not specific because it can be produced by tumors that partially occlude the aqueduct or fourth ventricle, such as pinealomas, astrocytomas, papillary ependymomas of the fourth ventricle, or posterior fossa arachnoiditis. When we find this, we therefore, feel that it is indicative of cysticercosis only, when there is additional data to support the diagnosis, such as x-ray calcifications elsewhere in the brain, cysticerci in the muscles or mononuclear cells in the cerebrospinal fluid.

In the management of cysticercosis we prefer to put those patients who do not show increased intracranial pressure (the intraparenchymatous group) on anticonvulsant therapy, especially if the x-rays demonstrate many calcifications. If there is only one area of calcification, we allow sufficient time to permit the pathological process to settle and calcification to take place in any other possible lesions, in order to be certain that we are dealing with only one cyst. If, after two years, the x-rays do not reveal additional calcifications, if the electroencephalogram and clinical findings are consistent with the same localization, and finally if seizures cannot be controlled by anticonvulsant medication, we then attempt surgery. In the difficult cases where there are many intracerebral calcifications and anticonvulsant therapy is unsuccessful, we try to localize the responsible focus by clinical findings and electroencephalogram, and if successful, submit the patient to surgery. In any case, where there is increased intracranial pressure, we, of course, undertake surgery. It is important to emphasize from the surgical aspect that the cyst must not be broken. Otherwise an arachnoiditis will result. Where there are multiple subarachnoid cysts we do not attempt removal, but rather perform an external shunt procedure.

DR. ROBERT S. SCHWAB: Recently when I was in Uruguay I saw an exhibit of hydatid cysts in the brain with their EEG findings. Their chief problem was that the large size of these space-occupying masses produced widespread electrical abnormality which sometimes confused the localization considerably. There was some discussion of other types of cysts in this exhibit. I do not remember whether Dr. White's cases have had EEG localizations or not.

Personality Correlates of Two Phases of Stress Reactions, D. H. Funkenstein, M.D., S. H. King, Ph.D. and M. E. Drolette, M.P.H.

One hundred and twenty-five healthy college students studied during laboratory stress experiments were scored on two aspects of their stress rections: 1. Their acute emergency reactions in terms of the immediate emotion expressed by them—anger directed outward, anger directed inward, or anxiety. 2. Their abilities to handle stress as time passed in terms of mastery or failure in mastery of stress. The Mastery Group was subdivided into a Master Group and a Delayed Master Group; the Failure in Mastery Group into an Unchanged Group and a Deteriorate Group.

The acute emergency reactions of the subjects were interpreted as representing the "structure" or "style" of the personality and the ability to handle stress as time passed, the "function" of the personality. No relationship was found between the form of the acute emergency reactions of the subjects and their abilities to master or failure to master stress as time passed.

Personality data were collected by psychiatric interviews, psychological tests and sociological questionnaires. The personality data which were related to the "inner" aspect of personality and therefore represented the personality "structure" or "style" correlated significantly with the acute emergency reactions but failed to correlate with the ability to handle stress as time passed. The personality data which were related to the "function" of the personality correlated significantly with the ability to handle stress as time passed but not with the acute emergency reactions.

Acute Emergency Reactions.—The personality data which correlated with the acute emergency reactions were the following:

1. *The perceptions of the parents*: The relative importance of one parent in relation to the other as the chief source of authority and affection and as the role model was determined. The men who reacted during acute stress with *anger directed outward* perceived their fathers as the chief sources of authority and as their role models and their mothers as their chief sources of affection. Their relationships with their fathers was poor. These were "traditional" families. The men who responded with *anger directed inward* also perceived their fathers as the chief sources of authority and as their role models with their mothers as the chief sources of affection. This group differed from the previous one in that the differences between the parents were not so marked. The relationship with father was good and father shared some of the authority with the mother, and mother some of the affection with father. This was a "Transitional" family. The men who responded with *anxiety* were "mother related" perceiving their mothers as their chief sources of authority and affection and as their role models.

2. *The Internal Concept of Self:* Using the Brownfain Concept of Self Test, the *anger directed outward* group showed a marked difference between the "best self" and the "worst self"; the *anxiety* group a small difference between the "best self" and the "Worst self" with the *anger directed inward* group in between the anger outward and anxiety groups on this measure.

3. *The fantasies as determined by Thematic Apperception Tests:* The *anger outward* group fantasied little aggression whose punishment was by men using verbal and physical means. Its consequences were not calamitous to them. The *anger inward* group fantasied much aggression followed by punishment and guilt. Punishment was usually at the hands of a woman using the threat of withdrawal of love. The anxiety group fantasied that aggression was often followed by injury at the hands of a woman who also used the threat of loss of love as a means of punishment.

4. *The Social Attitudes and Social Class:* The *anger inward* group differed from the other students in being predominantly fifth generation Americans. The *anger directed outward* group was higher on social attitude scales such as those used in The Authoritarian Personality than were the *anger directed inward* or *anxiety* groups.

The Ability to Handle Stress as Time Passed.—The personality data which correlated with the ability to handle stress as time passed were the following:

1. *Measures of Interpersonal Relations:*

The Master Group felt that others felt the same about them as they felt about others. The Delayed Master Group felt that others thought more of them than they did of themselves and the Deteriorate Group that others thought less of them than they thought of themselves.

2. *The Ability to Assess Reality:*

The Master and Delayed Master Groups assessed the reality of their performances during the stress experiments significantly better than the Unchanged and Deteriorate Groups.

3. *The Integration of the Personality:*

A Phrase Association Interview technique developed by Ravven was used. Integration was defined in relation to the balance between certain characteristics of emotions expressed by the subjects. According to these criteria, the Master Group showed much better integrated personalities than did the Unchanged and Deteriorate Groups. These latter groups showed different imbalances between the emotional factors.

Working Hypotheses.—The working hypotheses based on this work and other work of ours are: the acute emergency reactions represent one aspect of personality, namely *structure*, and are predictive of the emotional content of a psychosis or psychosomatic illness should one develop; the ability to handle stress as time passes represents a different aspect of personality, namely *function*, and is closely related to proneness to develop a psychosis or a psychosomatic illness.

Discussion.—Dr. Erich Lindemann: Thank you for an overwhelming amount of data brought together in a remarkably appealing way in these slides. Obviously a lot of work has been going on in the area of conceptual scheme and the correlations. It is quite a problem to pick out a point at which to start. I might ask a simple question. How did you motivate these boys to stick at it that long? How can you compare these boys with psychotic patients whose motivational set is so different?

Dr. William H. Sweet: I should like to know more about the test procedure.

DR. FUNKENSTEIN: The question concerning the method used to motivate the students was an extremely important one. Without adequate motivation there can be no stress experiments. We put a great deal of thought into this problem in planning the project. We represented to them the authority of the college, especially since we had the cooperation of some college authorities in obtaining the subjects. In addition, we personally talked to every man prior to the start of the project explaining its purpose. Fifteen minutes of each testing period was set aside for "passing the time of day" with the student in order to build a relationship. Again the college newspaper was read so that if a student won an honor he could be congratulated on it when he next came to a testing session. All of this contributed to the building of a positive relationship between the personnel working on the project and the subjects.

The psychotic patients have not been studied in a stressful situations. We imply that psychotic patients are under constant stress whose stimulus is within their own personalities. Students during stressful situations will show similar emotional and physiological reactions to those manifest by psychotic patients during non-stressful situations. During non-stressful situations, students do not show reactions similar to psychotic patients.

The method for inducing stress was by using an apparatus we termed a "sonic confuser". The subject speaks into a microphone and hears his own voice .2 second later through a headset. Probably due to inference with the "feedback" of the voice, stammering and other difficulties in speech are induced. Using the "sonic confuser" the student was asked to repeat a "culture-free" story from memory as rapidly as possible. This resulted in difficulty in speech and marked frustration with an accompanying emotional reaction.

RECOVERY FROM BRAIN OPERATION. HANNIBAL HAMLIN, M.D.

The recuperative properties of the human brain are seldom brought to us by the thoughts and feelings of the patient himself. A case history is presented of a newsman who underwent a negative exploratory craniotomy under local anesthesia on suspicion of tumor of the left hemisphere. Being a converted left-hander he developed transient aphasia and apraxia along with hallucinatory and depersonalization experiences.

During his convalescence he was persuaded to write about his reactions and attitudes during the acute recovery period, partly as a rehabilitation device and also in expectation of extracting information of neuropsychologic interest from a person possessing unusual insight and expository ability. The literary product of his recall concerning the manifold difficulties of his early postoperative experience proved to be a cogent and witty revelation of the belaboured notion that the dysphasic is intellectually aware of much more than the casual observer may realize.

Discussion.—DR. ERICH LINDEMANN: Thank you for this report. It is a story which we rarely hear from patients—the reactions of his total personality to his defect, his frustration and feelings, and way of feeling and experiencing what he calls hallucinations.

DR. WILLIAM H. SWEET: Dr. Hamlin and I were entranced by this man's capacity to analyze and express his feelings. We have had occasions since to guide ourselves and others in recoveries. Another patient had hallucinations: although he weighed 200 pounds, he felt he had diminished to the size of a Tom Thumb. He was terribly afraid that someone would throw him away with the laundry. Some people made fun of this. His resentment was extreme. Later on when he had recovered and lost his hallucinations, he still had resentment for people who had not been sympathetic with him. The account given by Dr. Hamlin makes it clear what an interesting task we have, not only to appraise the unreal situation of the patient, but also to help him through it.

DR. WILFRED BLOOMBERG: While it is true that this was a very articulate patient who could say how he felt, we should give credit to Dr. Hamlin for having asked him. Too often, we just don't ask, and don't listen. If there were more people like him among doctors, it might not have been necessary to invent the word "psychosomatic".

DR. LINDEMANN: From the point of view of investigative work with forms of sensory disturbance, especially with the whole area of hallucinations, there might be enormous opportunities with patients like this. One is reminded of the kind of sensory disturbance of patients under mescalin and lysergic acid. They also have a disturbance in the perception of size.

BOOK NOTES AND REVIEWS

Books received are acknowledged and given a brief description in this section. Selections serving a variety of interests will be made from these for a more extensive review as far as space will allow.

BOOK REVIEWS

Naumburg, Margaret. PSYCHONEUROTIC ART: ITS FUNCTION IN PSYCHOTHERAPY. (New York: Grune & Stratton, 1953. Pp. x + 148.)

In his preface to this book, Kenneth E. Appel gives a succinct summary and evaluation ot it: "Miss Naumburg's book is a study and record of spontaneous art productions in psychotherapy. Painting and drawing are encouraged and used as levers of psychotherapy. This book represents a challenge to every psychiatrist practicing psychotherapy. It is an expansion of Miss Naumburg's previous work. A relatively new method of psychotherapy is employed in a severe psychiatric condition, with markedly beneficial results. Miss Naumburg's use of graphic illustration is not to be confused with the use of the Rorschach figures and test. The former is therapeutic, the latter diagnostic. A clear account of the difference is given." One chapter was contributed by A. G. Woltmann, who administered the psychological tests and the three Rorschach examinations at various stages of the psychotherapy. Woltmann gives a "correlation of the patient's, Rorschach and other tests with the patient's art productions". Another chapter, including a partial "blind" analysis of the patient's first Rorschach record, was written by Z. A. Piotrowski, who offers "comments on the Rorschach test and its relation to the psychotherapeutic use of graphic art". The book is profusely illustrated with 65 reproductions of the patient's graphic works, some of them in chromatic colors.

After an introduction discussing art therapy in theory and practice, Miss Naumburg describes the three year therapy of a young unmarried woman who produced 450 color paintings and about 200 crayon drawings. The patient preferred to make her pictures at home, bringing them to the therapy sessions. There were 125 therapy sessions in all, varying in length from two to two and a half hours. The therapist's chief function was to help the patient interpret her own graphic productions. The therapy was quite successful. There is a good follow-up for the two and a half years subsequent to the termination of treatment. The interesting point is that this patient, as well as nearly all patients whom Miss Naumburg reported as having improved through art therapy, were obsessives. Could this form of therapy be the most fruitful technique in the treatment of obsessive neurotics, who are difficult therapeutic cases? The book gives a tentative theoretical answer to this question. It does not deal with abstract art but indirectly and implicitly it contributes to an understanding of "non-objective art". Some thirty years ago Nolan Lewis used the patient's spontaneous graphic productions as an essential adjunct to psychotherapy. Miss Naumburg made them the primary therapeutic agent. Her "Psychoneurotic Art" is very competently written, giving a concise but sufficiently detailed account of the therapeutic process and its results. This therapeutic procedure is indeed a challenge to all psychotherapists and it may become very helpful.

Johnson, Donald M. THE PSYCHOLOGY OF THOUGHT AND JUDGMENT. (New York: Harper & Bros., 1955. Pp. x + 515. $6.00)

This is a collection of summaries of a vast number of articles and books dealing with mental processes involving thought and judgment. The author is restrained in his generalizations which are limited essentially to the obvious descriptions of stages of problem solving and the influence of various conditions on the efficiency and results of solving problems

of a manual or abstract nature. The book is largely a description of experimental procedures used by psychologists and of the results obtained with these procedures. Since it is extremely difficult to devise experiments on creative thinking, the book contains very little on this topic. In fact, creative thinking is believed to differ but in degree from reproductive thinking or drudgery. The book is of greatest value to those who contemplate similar experiments. In this very difficult field science has not given us new insights or new discoveries. The study of thought and judgment is still in the phase of trying to devise methods which would demonstrate certain well known facts in an experimental setting and thus provide a well controlled proof of the validity of a number of generalizations. Among these generalizations are statements like these: that training improves performance; that once a successful mental procedure has been mastered to solve problems, there is a tendency to continue using it even if a simpler and easier procedure is available; the tendency is to perseverate with the more difficult and complicated but familiar procedure instead of shifting to a simpler and shorter but novel procedure. Group discussions tend to be more rigid and less productive than solitary thinking or an exchange of ideas between two, or at most several persons. It can now be demonstrated experimentally that the majority of people are intellectually inhibited when they have to think about new problems out loud in a group. In practically all of the studies reported, the emphasis is on achievement rather than on the thought processes used in arriving at the results. The book is not organized around some basic problems or issues. The presentation is unsystematic and frequently it is hard to see the forest for the trees. However, it is a good survey of what has been done in the field experimentally and is good reading for those who wish to investigate the problems further. The reader who would like to get information about the biological function of thinking, the psychopathology of thinking, the sense of reality, or the processes of validating generalizations, will not find it in this book. It was not the author's purpose to deal with these most complex matters. His book contains a great deal of information about the subject as seen from one particular point of view, the experimentalist in a university laboratory. Valuable suggestions for further reading are found in the extensive bibliography.

Z. A. PIOTROWSKI

Patterson, C. H. THE WECHSLER-BELLEVUE SCALES: A GUIDE FOR COUNSELORS. (Springfield Ill.: C. C. Thomas, 1953. Pp. VIII + 146. $3.75)

The primary purpose of the Wechsler-Bellevue intelligence scales is to measure general intelligence. The adult content of the test questions and the brevity of the scales established the Weschsler-Bellevue as the main adult test used at present. However, the test is being used as an aid in personality analysis and diagnosis with the encouragement and active participation of its originator, David Weschsler. In his very lucid, brief, and thorough review of 250 pertinent publications, Patterson discusses the published results of the various applications of the Bellevue tests. The tests have a high degree of reliability and good validity as measured by their correlations with other intelligence tests. Patterson found that certain abbreviated forms of the tests are as valid as the complete tests scale. However, he concludes that the Bellevue tests are not valid at all as measures of mental deterioration and of vocational aptitudes. Nor are they dependable as aids in neuropsychiatric diagnosis. The Bellevue tests perform well their primary function as measures of general intelligence but they fail in the other more difficult tasks which have been imposed upon them. Nevertheless, most clinical psychologists continue looking for diagnostic cues and evidence of personality traits in the Bellevue test data. This seems unwarranted but is justified by the fact that, while few reliable differences have been found between group average scores on the sub-tests of healthy subjects and group average scores of any patient group, a small percentage of schizophrenic and organic cerebral cases produce characteristic test patterns. Nearly all of these cases are not borderline or mild but definite, advanced cases. Practically all psychologists who engage in research try to establish similarities and differences between

all the normals and all the patients. They do not aim at finding pathognomonic signs which occur only in a minority of patients. Such pathognomonic test signs can be helpful, if valid, even though they cannot be applied to every case. A vocational counselor is interested primarily in the ability of a patient to perform some useful work regardless of the diagnosis. Patterson's survey of literature shows that no studies were made in which the verbal intelligence quotient and the chronological age were kept constant, i.e., in which the comparison of test performance was limited to individuals of about the same age and about the same verbal intellectual level. About twenty years ago it was found that on the more homogeneous Stanford-Binet intelligence tests, the differences between psychoneurotics and schizophrenics increased when both groups were subdivided into three groups: with a low, a medium, and a high mental age. It is possible, therefore, that the more heterogeneous Bellevue tests would differentiate among different patient groups better if patient groups of the same verbal intelligence and age levels were compared with one another. The relative difficulty of a test within a battery of tests seems to vary with the intellectual level of the subject. About a quarter of a century ago, before the modern personality tests have become popular, clinical psychologists came to the conclusion that a marked discrepancy between the level of performance on purely verbal tests and the level of performance on non-verbal, performance tests was an approximate measure of intellectual inefficiency. The degree of this discrepancy was not associated with any particular cause which could be psychogenic and reversible, or organogenic and irreversible, or both. Counselors might be interested in a simple measure of intellectual inefficiency of this sort if its validity were established. Patterson, a Veterans Administration psychologist, made a valuable contribution not only by his conscientious review of the literature but by the systematic and logical arrangement of the problems which have been investigated and published. His conclusions are fully warranted by the available results reported in print. It seems, however, that not all of the crucial problems were formulated and investigated. Among the insufficiently examined procedures, in Wechsler's combining of the verbal and much less variable, as well as the non-verbal and much more variable, intelligence tests performances when computing the intelligence quotient. It cannot be said, therefore, that the last word has been said about the use of the Bellevue intelligence test scales as aids in diagnosis and personality analysis.

ZYGMUNT A. PIOTROWSKI

Szondi, L. TRIEBPATHOLOGIE (PATHOLOGY OF DRIVES). Vol. I. (Bern, Switzerland: Hans Huber Verlag, 1952. Pp. 543.)

This is the author's third large volume describing his remarkable and daring psychological test and its application in psychopathology. The test consists of six sets of eight photographs; one set is shown to the subject at a time with the request to pick out the two persons liked the most and the two persons disliked the most. At the end of each examination there is a list of 12 likes and 12 dislikes. The photographs present faces of patients. In each set there is a picture of a sadist (man who killed the woman he loved), of a male homosexual, of an epileptic, of a hysteric, of a catatonic and of a paranoid schizophrenic, of a depressed and of a manic manic-depressive psychotic. The assumption made by Szondi is that each of these patients suffers from the exaggerated development of a psychological need or drive which is universal. Another assumption implies that any individual (ill or healthy), who is troubled by insufficient gratification of one of the eight basic needs or by repression of any of these needs, responds immediately and unconsciously to those photographed patients who had very marked difficulties with the same basic drives. Thus a person who is tense because of difficulties with his need for exhibitionism and for being admired is expected to respond to the hysterics. If he likes them, he is said to accept his exhibitionism unconsciously but suffers from insufficient gratification of this drive. If he dislikes the hysterics, he is said to repress his exhibitionistic drive. If he both likes and dislikes them, he is described as being ambivalent about his exhibitionism. If he shows no interest in the hysterics, he is thought to have no

tension in his drive for recognition or exhibitionism either because he has satisfied the need fully or because this need is so small that satisfaction is easily obtained. Homosexuals are used to measure the need for tenderness in psychosexual relationships, and sadists to measure the need for active and forceful initiative in sexual matters. It goes without saying that the like for, or dislike of, certain types of photographed patients, does not imply that the subject is suffering from the same illness or builds defenses against the illness characterizing the photographed patients. The test can be very complex. Since there are eight drives and four possible attitudes toward each of them, we obtain 32 different attitudes. The single drives can be combined with others in various patterns, allowing for thousands of various combinations. The point of good research is to concentrate on the most frequent and the most meaningful combinations. The main difference among Szondi's three books consists of improvement in just that direction: emphasis on the more frequent and more significant patterns and the gradually more intensive psychological analysis of the implications of these patterns.

The test is easy to administer and takes no more than ten minutes. A single examination can be used but it is psychologically much more profitable to obtain a series of test records, at least six; Szondi suggests that ten examinations be made within a relatively short time. While the technique of administration and scoring is simplicity itself, the psychological analysis of the data requires a very thorough and critical study of Szondi's vast system of personology. It is very easy to misuse the test. Szondi's style is literary which impresses some readers as being vague. Actually he is lucid but complex. It is a bold idea to use the simple technique of asking people for their likes and dislikes when shown sets of photographed faces as a basis for far-reaching and significant psychological and psychopathological inferences. The argument that people are very poor judges of character when trying to read character from photographs does not apply here. In the test, the subject is not required to describe the personality traits of the patients he sees on the photographs. He is asked merely to indicate his potential action: whether he likes or dislikes, whether he would like to be in the company of the patient or would prefer to avoid him. He is not told who the photographed individuals are. Szondi's views of the structure and etiology of the human personality are also novel and complex though lucid. He attempts to reconcile in his ambitious system heredity, psychoanalysis and general psychology. This book is a great advance over the two former volumes. The previous book describing the test will be needed to make the understanding of this one complete. Much time will be needed before the original ideas of Szondi will become known and understood, let alone accepted. The reviewer's limited experience with the test and exchange of ideas with colleagues who are using the test led him to believe that this is a valuable contribution to experimental personology but that the test must be known thoroughly to make it a fruitful addition to our clinical psychology armamentarium. It is relatively easy to make comparisons between one state of a subject and another intra-personal change in the same subject. It is far more difficult to compare different individuals using this test. Those who want to make inter-personal comparisons will find this test more difficult than those whose primary interest is to employ it as an aid in measuring intra-personal changes in the basic drive structure. Whatever the final judgment of science will be, there is no doubt that Szondi's achievement is most significant for the experimental study of personality. Once the test is well validated and well understood, it may become the simplest and quickest method of assessing important personality traits.

Z. A. PIOTROWSKI

Arnold, O. H. SCHIZOPHRENER PROZESS UND SCHIZOPHRENE SYMPTOMGESETZE: EINE PROGNOSTISCHE-STATISTISCHE GRUNDLAGENSTUDIE. (Vienna: W. M. Maudrich Verlag, 1955, Pp. 86. $3.75)

This monograph on the schizophrenic disease process and the laws of symptom formation in schizophrenia, with an emphasis on prognosis, was written by a neuropsychiatric assistant of the University Clinic in Vienna. The statistical surveys, summarized in sixteen tables were based on 500 consecutive schizophrenic cases treated in the post-war

period. The first chapter reviews the theories of schizophrenia briefly and incompletely. With but few exceptions this review is limited to German and Austrian literature. The theories are evaluated without giving the reasons for the judgment. Eugene Bleuler's concept of schizophrenia is rejected as a backward step. The author approves Stransky's definition of schizophrenia as intrapsychic ataxia and subscribes to C. Schneider's formulation of schizophrenia and of its etiology. He assumes that schizophrenia develops when the personality is both specific and constitutionally weak, and when the patient is exposed to great stresses. Schizophrenia is a mental illness characterized by marked disturbances in the steadiness and fluency of vital energies. These disturbances, in turn, cause uncontrollable changes in the direction of energy. The author preferred to deal with many clinically definable symptoms instead of relying on global personality syntheses. This emphasis on definite clinical symptoms allowed a statistical treatment of his observations. The frequency of about 50 symptoms was calculated in the distinguishable phases of schizophrenia from incipient to fully developed forms. A five degree scale of personality defect was developed in terms of clinical psychiatric symptoms. It was applied to all of the 500 patients. Moreover, the patients, all schizophrenic, were divided into five groups as follows: (1) complete remission; (2) working and supporting himself; (3) left hospital but requires care at home; (4) alternating care at home with care in the hospital; (5) in need of continuous hospitalization and incapable of staying at home. The Pearson correlation between the degree of defect as measured by the above symptom scale and the severity of schizophrenia, as measured by the five patient groups, from the least to the most debilitated, was .70 with a very low standard deviation of .0007. The correlation is significant.

The author found that shock treatments benefitted most those patients who exhibited great variations in energy output, whose energy output was unsteady and level of adjustment variable. The author stresses the point that the validity of his and any prognostic criteria depends on the time at which the prognosis is made. The prognostic criteria was evolved from an analysis of the frequency with which the symptoms, constituting the criterion, appeared in the different patient groups. They were checked also on a new group of 20 additional cases. Arnold did not divide his patients into the customary categories of catatonic, paranoid, hebephrenic and simple schizophrenia. The probable reason for this may be that most of his patients were early ambulatory cases in whom the differentiation among the traditional four subgroups cannot be made with a satisfactory degree of validity. Arnold states that his prognostic criteria proved the more valuable, the more definite and unquestionable the diagnosis was. Among his patients, only 24% were hospitalized while 19% were believed to have had full remissions. The clinical symptoms which point to a good prognosis include disturbances of voluntary attention; loss of directed and logical thinking with involuntary shifting of thought; condensations; blocking; religious and cosmic experiences; quick and jumpy thinking; vague and unsystematized delusions. The symptoms which indicate a poor prognosis imply that the patient suffers from a firm delusion that his thoughts and actions are manipulated by someone else without or against his will. The feeling of having lost the power of doing anything on his own initiative is also unfavorable prognostically, if one excludes from it the subjective "experience of hearing one's own thoughts". A less decisive negative prognostic significance have disturbances of vital feelings, parakinesis, parabulia and negativistic stupor.

Psychiatric contributions based on so large a case material as Arnold's are very rare. The author's empirical findings can be accepted whether the reader does or does not share his theory of schizophrenia in all details or not at all. The factual tabulated data are empirical and are repeatable. They do not lead to any new theoretical concepts of schizophrenia but are a definite contribution to clinical psychiatry. They have been inspired by a much needed desire to check the claims made regarding the prognosis of schizophrenia against a large number of clinical observations.

Z. A. PIOTROWSKI

Obituaries

Joseph C. Yaskin M. D. (1891-1955)

Dr. Joseph Yaskin, professor and head of the neurological division of the Graduate School of Medicine of the University of Pennsylvania, died on August 11, 1955 at the age of 64 years. He was born in Odessa, Russia, came early to America and obtained his medical degree at the old Medico-Chirurgical College in 1914 with high honors.

Dr. Yaskin was very active professionally, was associated with Philadelphia hospitals in the field of neurology for 35 years and contributed a large number of papers on neurology and psychiatry. He was especially well known as an able, inspring teacher. He served as an officer in the Army Medical Corps during World War I, and worked in the Selective Service during World War II. He will be long remembered by his friends and associates who were privileged to know and work with him intimately.

Carl Dudley Camp, M. D. (1880-1955)

Dr. Carl Dudley Camp, Professor Emeritus of Neurology at the University of Michigan Medical School, died in Ann Arbor on May 23, at the age of seventy-four years. Dr. Camp was born in South Watauga, Tennessee, on October 2, 1880. His early education was by private tutors. He studied medicine at the University of Pennsylvania, from which he was graduated in 1902. After completing his internship at Howard Hospital in Philadelphia, he received training in neurology under the late William G. Spiller. He served as Instructor in Neurology and Neuropathology at the University of Pennsylvania and as Assistant Neurologist at the Philadelphia General, Philadelphia Polyclinic and University of Pennsylvania Hospitals from 1904 to 1907.

In 1907 Dr. Camp joined the faculty of the University of Michigan Medical School as Clinical Professor of Diseases of the Nervous System. In 1920 his title was changed to that of Professor of Neurology. He retired from his academic position in 1950, but continued his specialty until shortly before his death.

Dr. Camp served for many years as a Collaborating Editor of the Journal of Nervous and Mental Disease. He became a member of the American Neurological Association in 1906, and was made a senior member in 1945; he served as Vice-President in 1915 and again in 1918. He was one of the original members of the Central Neuropsychiatric Association. He was president of the Detroit Neurological Society (now the Michigan Society of Neurology and Psychiatry) in 1914. He was also a Fellow of the American Medical Association and a member of the Philadelphia Neurological Society, the American Association of Pathologists and Bacteriologists, the American Psychopathological Association, the Research Club of the University of Michigan, Nu Sigma Nu, Alpha Omega Alpha, and Sigma Xi. He was an honorary member of the American Academy of Neurology.

Dr. Camp was very active in investigation and in writing during his earlier years, but his later years were devoted mainly to practice and teaching. Although he was eminent in the practice of his specialty, it is his stature as a teacher that will be most widely remembered. He greatly stimulated the men who worked with him, and many of those he trained have become leaders in the practice and teaching of neurology. In 1947, on the fortieth anniversary of his affiliation with the University of Michigan, his former students presented an oil portrait of him to the University.

Dr. Camp is survived by his wife, the former Joan Joy, whom he married in 1911, and by one son, Carl D. Camp, Jr.

RUSSEL N. DEJONG

NOTES AND NEWS

THE THIRTEENTH ANNUAL CONFERENCE OF THE AMERICAN GROUP PSYCHOTHERAPY ASSOCIATION, INC.

The Thirteenth Annual Conference of the American Group Psychotherapy Association will be held Friday and Saturday, January13th and 14th, 1956, at the Henry Hudson Hotel, 353 West 57th Street, New York City.

Six all-day workshops will meet from 9.00 A.M. to 12.00 M., and from 1.00 P.M. to 3.00 P.M., on the 13th of January. Special registration will be required for these workshops. Topics are: The Dynamics of Group Psychotherapy; The Role of the Therapist; Resistance in Group Psychotherapy; Parallel Individual and Group Psychotherapy; Counterindications for Group Psychotherapy, and Research. Among the leaders of these workshops are: Dr. Lewis H. Loeser, Dr. Helen Durkin, Dr. Maurice E. Linden and Dr. Jerome D. Frank.

Three scientific sessions will he held, beginning Friday, at 3.30 P.M., through Saturday, 5.30 P.M.

For the morning of Saturday, January 14th, six round tables have been arranged. The subjects of these are: differential Levels of Group Treatment of Parents of Children; Special Problems in Group Therapy; Total Group Treatment in Mental Hospitals; Group Psychotherapy with Children; Group Psychotherapy with Ambulatory Schizophrenic and Borderline Cases and a tape recording of a session of an analytic group. Films dealing with group psychotherapy will also be shown.

Inquire at the Association's office, 228 East 19th Street, New York 3, N. Y.

DR. HYMAN PLEASURE APPOINTED DIRECTOR OF MIDDLETOWN STATE HOMEOPATHIC HOSPITAL

Appointment of Dr. Hyman Pleasure as director of Middletown State Homeopathic Hospital has been announced by Dr. Paul H. Hoch, Commissioner of Mental Hygiene. Dr. Pleasure will succeed Dr. Walter A. Schmitz who retires as director on July 31 after 41 years on the staff of the hospital.

Prior to his appointment at Middletown, Dr. Pleasure served for the past six years as assistant director at the Edgewood Division of Pilgrim State Hospital in Brentwood, L. I. He began his state service in 1937 as an assistant physician at Dannemora State Hospital and four years later transferred to Central Islip State Hospital as senior assistant physician, advancing through various staff positions until November 1948 when he became assistant director (clinical) at Dannemora. He joined the staff at Pilgrim in March 1949.

PROFESSOR OF PSYCHIATRY APPOINTED AT CHICAGO

Dr. C. Knight Aldrich, associate professor of psychiatry in the University of Minnesota Medical School, has been appointed professor of psychiatry and chairman of the department of psychiatry in the University of Chicago School of Medicine, Dr. Lowell T. Coggeshall, dean of the University's division of biological sciences, announced today. Dr. Aldrich received his M.D. degree from Northwestern University in 1940, and served his interneship at Cook County Hospital. His chief fields of interest are teaching, both of medical students training to become psychiatrists and of those who will specialize in other branches of medicine, and in furthering integration of psychiatry with other medical specialties.

VALUE OF V.A. OUTPATIENT CLINICS

Mental hygiene clinics of Veterans Administrations are keeping an average of 2,200 mentally sick veterans out of hospital every year.

VA said a great number of these veterans are engaged in useful, productive work while receiving treatment and generally improve sufficiently to end their outpatient treatments.

The cost of treating these 2,200 in mental hygiene clinics an average of once a week for

one year is roughly $1,000,000. The cost of treating the same number of patients in a neuropsychiatric hospital is about $5,000,000 per year. VA therefore estimates that about $4,000,000 a year is saved in preventing hospitalization.

Only veterans with service-connected neuroses or psychoses may be treated in VA's mental hygiene clinics. The law does not permit the treatment of nonservice-connected cases on an outpatient basis.

CONNECTICUT POSTGRADUATE SEMINAR IN PSYCHIATRY AND NEUROLOGY

The Ninth Connecticut Postgraduate Seminar in Psychiatry and Neurology will begin its courses of lectures on September 26, 1955 and will continue through May 7, 1956.

From September 26, through December 5, 1955 sessions in clinical neurology, neuroroentgenology, electroencephalography, neuroanatomy, neurophysiology, and neuropathology will be held on Mondays and Wednesdays from 3:00 to 9:00 p.m. at Yale University School of Medicine, New Haven.

From January 9 through March 5, 1956 (Mondays) from 3:00 to 10:00 p.m. sessions in general psychiatry (psychopathology, clinical psychology, psychiatric syndromes, therapy, psychosomatic medicine, geriatric psychiatry and psychiatry and law) will be held at the Connecticut State Hospital, Middletown.

March 12 through April 16 (Mondays) from 6:30 to 9:45 p.m. a course in child psychiatry will be given, and April 23 through May 7, 1956 (Mondays) from 4:30 to 10:00 p.m. there will be a course in pediatric neurology, both at Yale University School of Medicine, New Haven.

There are no fees for the above courses. Copies of the program may be obtained from the Office of the Assistant Dean for Postgraduate Medical Education, Yale University School of Medicine, 333 Cedar Street, New Haven, Connecticut.

The Congress of the Society of German Neurologists and Psychiatrists will be held in Hamburg, September 19–22, 1955. The main subjects are: "Motility and Motoric Behavior," "The Training of the Neurologist". For information: Dr. Magun, Nervenklinik, Hamburg-Eppendorf.

Dr. R. W. Wartenberg, Professor of Neurology at the University of California School of Medicine, San Francisco, has been elected honorary member of the Serbian Medical Society in Belgrade.

THIRTEENTH ANNUAL MEETING OF PSYCHOSOMATIC SOCIETY

The American Psychosomatic Society will hold its Thirteenth Annual Meeting at the Sheraton Plaza Hotel in Boston on Saturday and Sunday, March 24 and 25, 1956.

The Program Committee would like to receive titles and abstracts of papers for consideration for the program no later than December 1, 1955. The time allotted for the presentation of each paper will be twenty minutes.

Abstracts should be submitted in sextuplicate, for the Program Committee's consideration, to the Chairman, at 551 Madison Avenue, New York 22, New York.

STANLEY COBB, M.D.
Chairman, Program Committee

SYMPOSIUM ON VITAMIN B_{12}

Plans are being made for the first European symposium on vitamin B_{12} to be held the last week of May, 1956, in Hamburg. It will be under the auspices of the physiological chemist Prof. Kühnau, and the internist Prof. Jores. The physiology and the clinical application of vitamin B_{12} in human and veterinary medicine will be discussed. For information write to Doz. Dr. H. Bauer, Nervenklinik, Hamburg-Eppendorf, Germany.

NORTH SHORE HEALTH RESORT LECTURES

The second lecture in the Sixth Annual North Shore Health Resort Lecture Series on

Psychiatric Problems in Medical Practice will be given at 8:00 P.M., November 2, 1955 at the hospital. *The Use and Abuse of Sedatives and Stimulants* will be discussed by FRANCIS J. GERTY, M.D., Professor and Head, Department of Psychiatry, Director Psychiatric Division, Neuropsychiatric Institute, University of Illinois, College of Medicine; Senior Attending Psychiatrist, St. Luke's Hospital; Chairman Deans' Subcommittee on Psychiatry, V.A.H., Hines, Ill.

The third lecture in the Sixth Annual North Shore Health Resort Lecture Series on *Psychiatric Problems in Medical Practice* will be held on Wednesday evening, December 7, 1955, at 8:00 P.M. at the hospital, 225 Sheridan Road in Winnetka, Illinois. *The Management of the Anxious Patient* will be discussed by LEWIS L. ROBBINS, M.D., Director, Dept. of Adult Psychiatry, The Menninger Foundation, Topeka, Kansas.

The Commission on Education of the American Academy of General Practice has approved these lectures for informal credit. The J. B. Lippincott Company of Philadelphia will publish the entire series as a book. All royalties that will accrue have been assigned to the American Psychiatric Association.

The Index of Vol. 121 will appear in the August 1955 issue